UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION 5
230 SOUTH DEARBORN ST.
CHICAGO, ILLINOIS 60604

REPLY TO THE ATTENTION OF:

September 21, 1989 OHEA-5S

DRAFT

MEMORANDUM

SUBJECT: Evaluation of Disposal Options at the Chemical Waste Management Facility, Vickery, Ohio

FROM: J. Milton Clark, Director
Office of Health and Environmental Assessment

TO: David Ullrich, Director
Office of RCRA

Per your request, my office has had an opportunity to conduct a preliminary evaluation of the risk assessment and risk management disposal issues concerning the Chemical Waste Disposal Facility in Vickery, Ohio. Your office requested an evaluation of potential risks if the waste pile remained until an alternative disposal method, incineration, could be implemented and completed. The estimated time for incineration to be completed was 7 1/2 years. It should be noted that a complete risk evaluation is not possible because detailed information is not available on (1) chemical composition of wastes following fixation, (2) chemicals in ambient air (on-site and off-site) and (3) surface leachate run-off and leachate migration into ground water.

However, even with data limitations, OHEA believes the preferable remedy would be land disposal rather than incineration. This conclusion is based on the potentially very significant risks, to human health which would be incurred by the residential population over the estimated 7 1/2 years required for incineration. These risks are minimized, if not eliminated, by employing instead a more environmentally protective solution, RCRA landfilling, which is immediately available. Support for this conclusion is discussed below.

The presence of volatile organics in wastes before fixation were estimated to result in airborne, total VOCs of 230 ug/m^3 at 1 km to 69 ug/m^3 at 5 km (Clemments Associates page 43, 1983). Of these concentrations, about 38% would consist of methylene chloride, 28% chloromethane, 27% chlorobenze, and 4% chloroform. Lifetime (70 years) cancer risk for only methylene chloride at a distance of 1 km would be 4×10^{-3} and 1×10^{-4} at 5 km for an inhalation rate of $33m^3$/day. During the process of fixation with lime and cement kiln dust, some unknown portion of the total VOC mass present in the wastes escaped. Assuming even a high figure of 50% loss during fixation, the lifetime risks are still high.

Physical fixation or plastic covering would have an insignificant impact on reducing the rate of VOC release, particularly for compounds of small molecular weight, such as methylene chloride. In the absence of air data and assuming that the material at the site remained for 7 years before incineration was complete with half the VOCs lost during fixation, air inhalation risks could be 2×10^{-4} at 1 km and 5×10^{-6} at 5 km. Such residential air cancer risks would not be acceptable. Again, it must be noted that these values are crude, theoretical values. Air data would need to be collected for VOCs and PCBs for both the vapor and respirable particulate phase to more accurately estimate risks.

Quantitative estimates do exist regarding leachate composition and volume (CWM Technical Evaluation of September 12, 1984). With the stabilization systems used by Chemical Waste Management, leachate was shown through lab tests to have VOCs ranging from 9 ppb to 27 ppm, or total VOCs of about 36 ppm. Methylene chloride averaged 1.4 ppm in leachate. It was estimated that from 155,000 to 1,000,000 gallons of leachate would be released during compression and consolation of the stabilized material while in the landfill, assuming a depth of 45 feet. Since current storage conditions are similar to landfilling it is reasonable to assume that leachate is being generated. Over a course of several years, and with some contributions from rainfall due to permeation of the temporary cover, leachate generation could be considerable and approach or exceed that estimated from laboratory compression tests. The VOCs in the leachate would be expected to volatilize quickly and possibly contribute to ambient air levels of VOCs on-site and off-site.

Ground water contamination of VOCs have already been noted in the area (EPA-700/8-88-044). Several monitoring wells were found to have methylene chloride contamination up to 53 ppb. Continued storage at the site with no liner would be expected to significantly increase the potential for further ground water contamination. Surface water could also be affected from the runoff of leachate.

Recommendation

While fully quantitative estimates of risk cannot be made, the long-term (7 1/2 year) temporary storage of wastes at the site while incineration is being performed has a significant potential to cause undesirable and unnecessary risks to human health and environment. Because of these significant potential risks, and the fact that an immediate remedy is available in the form of land disposal in a RCRA landfill, OHEA encourages the land disposal option rather than incineration in this case.

cc. B. Muno
K. Bremer
J. Saric

Lockheed Engineering and Management Services Company, Inc.

CONFERENCE CALL FOR

CASE M-2363HQ, SITE 57 CHEM. WASTE MANAGEMENT

THURSDAY AUGUST 6, 1987

2:30 EASTERN TIME

202-382-7319

Lockheed Engineering and Management Services Company

Environmental Programs Office
1050 E. Flamingo Road, Suite 120, Las Vegas, Nevada 89119
(702) 734-3200

August 5, 1987

United States Environmental Protection Agency
Office of Solid Waste
401 M. Street SW
Washington, D.C. 20460

Attention: Mr. Rich Steimle
Via: K.S. Kumar 08/05/87
Subject: The Assessment of the Usability of the Data Generated for Case M-2363HQ, Site #57, Chemical Waste Management; Vickory, OH

Dear Mr. Steimle:

Enclosed are the inorganic, organic, and dioxin/furan data usability audit reports, and the quality control data evaluation report for case M-2363HQ site #57, Chemical Waste Management (CWM); Vickory, OH. This report is prepared for the Ground-Water Monitoring Task Force (GWMTF).

This report will be distributed to the members of the U.S. Environmental Protection Agency (EPA) Ground-Water Monitoring Task Force and their designees.

If you have any questions or comments, please do not hesitate to call. I can be reached at (702) 734-3350.

Very truly yours,

Earl S Moore for EWSmith

E. W. Smith
Senior Associate Scientist
Laboratory Performance
Monitoring Group

EWS/DLB:ahh

cc: Dr. Paul Friedman
Mr. Ken Partymiller
Mr. John Moore
Regional Representatives

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LIST OF ACRONYMS

BNA	Base/Neutral/Acid Fractions
CCS	Contract Compliance Screening
CLP	Contract Laboratory Program
CRDL	Contract Required Detection Limit
DL	Detection Limit
D	Dissolved Metal
DQO	Data Quality Objective
EPA	Environmental Protection Agency
IDL	Instrument Detection Limit
LCS	Laboratory Control Sample
PCDD	Polychlorinated dibenzo-dioxin
PCDF	Polychlorinated dibenzofuran
PE	Performance Evaluation
POC	Purgeable Organic Carbon
POX	Purgeable Organic Halide
QC	Quality Control
RPD	Relative Percent Difference
SMO	Sample Management Office
T	Total Metal
TOC	Total Organic Carbon
TOX	Total Organic Halide
VOA	Volatile Organic Analysis

1.0 INTRODUCTION

The U.S. Environmental Protection Agency (EPA) Hazardous Ground-Water Monitoring Task Force (GWMTF) is investigating 58 commercial land disposal facilities. The goal of the Task Force is to determine the extent of leakage of hazardous wastes (inorganic, organic, and dioxin/furan) into the ground-water.

The purpose of this report is twofold. It evaluates the accuracy and precision of the data generated by the laboratories, and also assesses the usability of the data (i.e., suspect, qualitative, semi-quantitative, or quantitative).

1.1 Sample Information

A total of 26 samples were collected at site #57, CWM, Vickory, OH. The following EPA sample numbers were assigned: MQB301-MQB326.

The traffic reports identified duplicates, blanks, and sample locations; there are 3 sampling blanks and 2 sets of duplicates. The duplicates and blanks are numbered as follows:

SAMPLING BLANK/DUPLICATE	EPA NUMBER
Trip Blank	MQB301
Field Blank	MQB304
Field Blank	MQB311
Duplicate	MQB314/MQB316
Duplicate	MQB307/MQB319

All samples are low concentration ground-water.

MQB306 and MQ326 are medium concentration leachate samples, and MQB310 is a medium concentration ground-water. Sample MQB313 is a low concentration surface water sample and the remaining samples are all low concentration ground-water.

2.0 EVALUATION MEASURES OF QC DATA

Accuracy, precision, blank contamination and detection limits are examined to insure compliance of laboratory procedures with the contract requirements and good laboratory practices.

2.1 Accuracy

The accuracy of the analytical methods is determined by the percent recovery of spiked compounds into the samples. Inorganic samples are spiked with known concentrations of analytes. Organic samples are spiked with known concentrations of surrogate compounds. The percent recovery of the spiked compound is calculated as:

$$(\%\ R)\ \text{Recovery} = \frac{\text{Observed concentration}}{\text{Spiked concentration}} \times 100$$

N.B.: for inorganic samples,

Observed conc. = spiked sample conc. - sample conc.

The accuracy, presented as %R $\pm$ standard deviation, must meet program data quality objectives (DQO) as outlined in Tables 2-1 and 2-2.

2.2 Precision

The precision of the inorganic analyses is determined by the relative percent difference (RPD) of each analyte in duplicate samples. The organic method, in comparison, uses matrix spike duplicate samples and surrogate compounds in the matrix spike samples to determine the RPD. The RPD is calculated as:

$$RPD = \frac{D_1 - D_2}{(D_1 + D_2)/2} \times 100$$

where:

RPD = Relative Percent Difference
D_1 = First Duplicate Value (percent recovery)
D_2 = Second Duplicate Value (percent recovery)

The precision, presented as RPD, must comply with the data quality objectives as outlined in Tables 2-3, 2-4, and 2-5.

LEMSCO has calculated the RPDs for the surrogate compounds in the matrix spike duplicate samples because the laboratory is not required to perform this task.

2.3 Blank Contamination

Laboratory and sample blank contamination is reported on Form III for inorganic data, and Form IV for organic data; the level of contamination must adhere to the contract conditions. If the contract limits are exceeded, the sample data must also be examined for the contamination and evaluated accordingly.

2.4 Reported Detection Limits

The estimated method detection limits are directly proportional to the concentration/dilution factor of the samples and the contract required detection limits (CRDL). For diluted samples, the estimated method detection limit is the CRDL times the concentration/dilution factor. For organic analyses, a new estimated method detection limit of greater than 10 times the CRDL may result in false negatives for some analytes. (The analytes may be present but not observed due to excessive dilutions.) Any changes in the instrument detection limits (IDL) for the inorganic analyses due to dilution of a sample will be reflected in the reported sample result.

TABLE 2-1 ACCURACY GOALS FOR INORGANIC AND INDICATOR ANALYSES(a)

Method or Parameter	Accuracy, %
Atomic Absorption method	75-125
Inductively Coupled Plasma method	75-125
Cyanide	75-125
Sulfate	80-120
pH	90-110
Total Organic Halide (TOX)	80-120
Purgeable Organic Halide (POX)	80-120
Total Organic Carbon (TOC)	80-120
Purgeable Organic Carbon (POC)	80-120
Chloride	90-110
Nitrate-Nitrogen	90-110
Phenol	80-120
Nitrite-Nitrogen	----
Bromide	----
Fluoride	----
Sulfide	----

(a) Source: Hazardous Waste Ground-Water Task Force Facility Assessment Program, Quality Assurance Plan.

TABLE 2-2 ACCURACY GOALS FOR ORGANIC SURROGATE AND MATRIX SPIKE COMPOUND RECOVERIES IN WATER SAMPLES[a]

Fraction	Compound	Accuracy (% Recovery)
VOA[b]	Toluene D8	88-110
VOA	4-Bromofluorobenzene (BFB)	86-115
VOA	1,2-Dichloroethane D4	76-114
VOA	1,1-Dichloroethene	61-145
VOA	Trichloroethene	71-120
VOA	Chlorobenzene	75-130
VOA	Toluene	76-125
VOA	Benzene	76-127
BN[c]	Nitrobenzene D5	35-114
BN	2-Fluorobiphenyl	43-116
BN	Terphenyl D14	33-141
BN	1,2,4-Trichlorobenzene	39-98
BN	Acenaphthene	46-118
BN	2,4-Dinitrotoluene	24-96
BN	Pyrene	26-127
BN	N-Nitroso-di-n-propylamine	41-116
BN	1,4-Dichlorobenzene	36-97
Acid	Phenol D5	10-94
Acid	2-Fluorophenol	21-100
Acid	2,4,6-Tribromophenol	10-123
Acid	Pentachlorophenol	9-103
Acid	Phenol	12-89
Acid	2-Chlorophenol	27-123
Acid	4-Chloro-3-methylphenol	23-97
Acid	4-Nitrophenol	10-80
Pest.[d]	Dibutylchlorendate	24-154
Pest.	Lindane	56-123
Pest.	Heptachlor	40-131
Pest.	Aldrin	40-120
Pest.	Dieldrin	52-126
Pest.	Endrin	56-121
Pest.	4-4'DDT	38-127

(a) Source: Hazardous Waste Ground-Water Task Force Facility Assessment Program, Quality Assurance Plan
(b) Volatile organics.
(c) Base/neutrals.
(d) Pesticides.

TABLE 2-3 PROGRAM PRECISION GOALS FOR INORGANIC AND INDICATOR ANALYSES[a]

Method or Parameter	Average RPD[b] Limits, ±%
Atomic Absorption method	20
Inductively Coupled Plasma method	20
Cyanide	20
Sulfate	20
pH	10
Total Organic Halide (TOX)	20
Purgeable Organic Halide (POX)	20
Total Organic Carbon (TOC)	10
Purgeable Organic Carbon (POC)	10
Chloride	10
Nitrate-Nitrogen	40
Phenol	20
Nitrite-Nitrogen	--
Bromide	--
Fluoride	--
Sulfide	--

(a) Source: Hazardous Waste Ground-Water Task Force Facility Assessment Program, Quality Assurance Plan

(b) Relative percent difference.

TABLE 2-4 PROGRAM PRECISION GOALS FOR ORGANIC MATRIX SPIKE/MATRIX SPIKE DUPLICATE ANALYSES (a)

Fraction	Compound	Average RPD(b) Limits, ±%.
VOA(c)	1,1-Dichloroethene	14
VOA	Trichloroethene	14
VOA	Chlorobenzene	13
VOA	Toluene	13
VOA	Benzene	11
B/N(d)	1,2,4-Trichlorobenzene	28
B/N	Acenaphthene	31
B/N	2,4-Dinitrotoluene	38
B/N	Pyrene	31
B/N	N-Nitroso-di-n-propylamine	38
B/N	1,4-Dichlorobenzene	28
Acid	Pentachlorophenol	50
Acid	Phenol	42
Acid	2-Chlorophenol	40
Acid	4-Chloro-3-methylphenol	42
Acid	4-Nitrophenol	50
Pest.(e)	Lindane	15
Pest.	Heptachlor	20
Pest.	Aldrin	22
Pest.	Dieldrin	18
Pest.	Endrin	21
Pest.	4-4'DDT	27

(a) Source: Hazardous Waste Ground-Water Task Force Facility Assessment Program, Quality Assurance Plan.
(b) Relative percent difference.
(c) Volatile organics.
(d) Base/neutrals.
(e) Pesticides.

TABLE 2-5 PROGRAM PRECISION GOALS FOR SURROGATE RECOVERY IN MATRIX SPIKE DUPLICATE SAMPLES[a]

Fraction	Average RPD[b] Limits, ±%
Volatile Organics	15
Base/Neutrals	50
Acids	40
Pesticides	30

(a) Source: Hazardous Waste Ground-Water Task Force Facility Assessment Program, Quality Assurance Plan

(b) Relative percent difference.

3.0 RESULTS OF LABORATORY QC DATA EVALUATION

3.1 Metals

3.1.1 Accuracy

Tables 3-1a through 3-1d summarize the accuracy performance for the metals based on the sample spike recoveries. These tables provide an estimate of the accuracy achieved for the metal parameters using average matrix spike recovery, standard deviation, and range. The average recoveries are within the DQO limits (Table 2-1) with the following exceptions:

Parameter	Level/Matrix	% R
Cd(T)	LW	137
Se(T)	LW	51
Fe(T)	LW	128
Mg(D)	LW	73
Cd(T)	MW	62
Se(T)	MW	20
Ag(T)	MW	72
Tl(T)	MW	74
Sn(T)	MW	64
Sb(D)	MW	37
Cd(D)	MW	68
Cu(D)	MW	53
Pb(D)	MW	0
Hg(D)	MW	60
Ag(D)	MW	62
Tl(D)	MW	0
Sn(D)	MW	34

Recoveries are not calculated if the sample results are greater than four times the spike added.

Tables 3-2a through 3-2d list the matrix spiked samples and the metal parameters with individual matrix spike recoveries outside the DQOs (Table 2-1). The individual recoveries are also summarized in the Inorganic Data Usability Audit Report.

3.1.2 Precision

Tables 3-3a through 3-3d summarize the precision performance for the duplicate metal analyses. All of the average RPDs are within the DQOs with the following exception:

Parameter	Level/M	RPD
Al(T)	LW	46
Se(T)	MW	65

The RPDs are not calculated if either one or both of the duplicate values are less than the CRDL.

The individual RPDs which are outside the precision DQOs are summarized in the Inorganic Data Usability Audit Report.

3.1.3 Completeness

The metal analyses were completed on all samples submitted to the laboratory.

3.1.4 Blank Contamination

Contamination was not reported for laboratory blanks but contamination was found in the sampling blanks. The contaminants and their concentrations are listed as follows:

Contaminant	CRDL (µg/L)	Conc.(µg/l)	Blank Sample ID/Type
Al(D)	200	259	MQB311/Field
Pb(D)	5	6.8	MQB311/Field
Pb(D)	5	16	MQB304/Field
Na(D)	5000	173000	MQB304/Field
Na(D)	5000	162000	MQB301/Trip
Na(T)	5000	160000	MQB301/Trip

3.1.5 Reported Detection Limits

The reported DL is the CRDL or a factor of the IDL for each metal parameter. A raised IDL can be caused by the sample dilution.

3.2 Inorganic and Indicator Parameters

3.2.1 Accuracy

Tables 3-4a and 3-4b summarize the accuracy performance for inorganic and indicator parameters based on the matrix spike recoveries. The average percent recoveries are within the accuracy DQO limits (Table 2-3) with the following exceptions:

Parameter	Level/Matrix	% R
$SO_4^{=}$	MW	70
SN^{-}	MW	13

Accuracy DQOs have not been established for bromide, nitrite, fluoride, and sulfide.

Individual matrix spike recoveries which are outside the accuracy DQOs are summarized in the Inorganic Data Usability Audit Report.

3.2.2 Precision

Tables 3-5a and 3-5b provide a summary of precision performance for inorganic and indicator parameters. Average RPDs for all parameters are within DQOs. The RPDs are not calculated if either one or both of the duplicate values are less than the CRDL. Precision DQOs have not been established for bromide nitrite, fluoride and sulfide.

Individual RPDs which are outside the precision DQOs are summarized in the Inorganic Data Usability Audit Report.

3.2.3 Completeness

Inorganic and indicator parameter analyses were performed on all samples as requested. Analyses were performed for bromide and nitrite on all samples, even though they were not required according to the traffic reports. These analyses were added by a contract modification.

3.2.4 Blank Contamination

Laboratory blank contamination was not reported but contamination was found in the sampling blanks. The contaminants and their concentrations are listed as follows:

Contaminant	CRDL (µg/l)	Conc(µg/l)	Blank Sample ID/Type
$SO_4^=$	1000	1880	MQB304/Field
S^-	1000	217000	MQB311/Field
POC	100	220	MQB311/Field
TOX	5	9.4	MQB304/Field

3.2.5 Reported Detection Limits

The reported DL is the CRDL or a factor of the IDL for each inorganic and indicator parameters. A raised IDL can be caused by the sample dilution. Contract required detection limits have not been established for bromide and nitrite.

3.3 Organics

3.3.1 Accuracy

3.3.1.1 Matrix Spike Recoveries

Table 3-6 summarizes accuracy performance for matrix spike compounds in the samples. These tables provide an estimate of the accuracy achieved for these compounds based on average percent recovery, standard deviation and range. All average recoveries are within DQO limits (Table 2-2) except 2-chlorophenol wich had an average recovery of 26.5%; QC limit 27-123%.

Individual matrix spike recoveries which are outside accuracy DQOs are summarized in the Organic Data Usability Audit Reports.

3.3.1.2 Surrogate Recoveries

Tables 3-7a thru 3-7c summarize the average accuracy performance for the recovery of surrogate spike compounds from samples, including matrix spike and matrix spike duplicate samples. All the average percent recoveries are within DQO limits (Table 2-2) except for 2-fluorobiphynyl in the sampling blanks, (Table 3-7c) and 2-fluorophenol in the matrix spike/matrix spike duplicate samples, (Table 3-7b).

Individual surrogate spike recoveries which are outside accuracy DQOs are summarized in the Organic Data Usability Audit Reports.

3.3.2 Precision

3.3.2.1 Matrix Spike/Matrix Spike Duplicates

Tables 3-8 summarizes precision performance based on matrix spike/matrix spike duplicate analyses for this case. All the average RPDs are inside the DQOs established for the program (Table 2-4).

Individual matrix spike RPDs which are outside precision DQOs are summarized in the Organic Data Usability Audit Reports.

3.3.2.2 Surrogate Spikes

Surrogate spike recoveries from matrix spike/matrix spike duplicate samples were used to calculate surrogate RPDs for comparison with Program DQOs (Table 2-5). Table 3-9 summarizes precision performance based on duplicate surrogate recoveries. All the average surrogate spike RPDs for this case meet the program DQOs.

3.3.3 Completeness

The required organic analyses were performed on all the samples submitted to the laboratory for this case except for chloroherbicide analysis on sample MQB325.

3.3.4 Blank Contamination

A summary of method blank contamination is reported in the Organic Data Usability Audit Report.

Contamination reported in the sampling blanks is listed in Table A2-1 of this report.

3.3.5 Reported Detection Limits

The method detection limits reported by the laboratory (reported as a value followed by a "U" on Form I for each parameter) were compared to the CRDL.

A summary of the method detection limits is reported in the Organic Data Usability Audit Report.

3.4 Dioxin/Furan Analysis

3.4.1 Accuracy

The accuracy performance in the native spike samples is summarized in Table 3-10. The recoveries are within the contract criterion of $\pm$ 40%.

3.4.2 Precision

Target analytes were not detected in the duplicate field and the laboratory duplicate samples so precision is not demonstrated.

3.4.3 Completeness

The required dioxin/furan analyses were performed on all the and the laboratory duplicate samples submitted to the laboratory for this case.

3.4.4 Blank Contamination

Dioxin/Furan contamination was not detected in the method blanks.

TABLE 3-1a ACCURACY PERFORMANCE FOR TOTAL METAL ANALYSES
IN LOW CONCENTRATION SAMPLES

Parameter	No. of Obs.	No. of Calc.	Average % Recovery	Std. Dev. % Recovery	Range % Recovery
Total Metals					
Aluminum	2	1	97	-	-
Antimony	2	2	96	4.0	92 - 100
Arsenic	2	2	100	27.6	72 - 127
Barium	2	2	96	1.0	95 - 97
Beryllium	2	2	99	1.0	98 - 100
Cadmium	2	2	137 *	41.1	96 - 178
Calcium	2	2	101	6.0	95 - 107
Chromium	2	2	98	0.0	-
Cobalt	2	2	93	2.5	90 - 95
Copper	2	2	105	1.0	104 - 106
Iron	2	1	99	-	-
Lead	2	2	125	27.1	98 - 152
Magnesium	2	2	82	11.5	70 - 93
Manganese	2	2	103	1.0	102 - 104
Mercury	2	2	105	5.0	100 - 110
Nickel	2	2	93	2.0	91 - 95
Potassium	2	2	95	0.0	-
Selenium	2	2	51 *	0.5	50 - 51
Silver	2	2	105	1.0	104 - 106
Sodium	2	2	105	1.0	104 - 106
Thallium	2	2	89	1.0	88 - 90
Tin	2	2	79	12.5	66 - 91
Vanadium	2	2	98	0.5	97 - 98
Zinc	2	2	99	2.0	97 - 101

* - outside DQO
- - not applicable

TABLE 3-1b ACCURACY PERFORMANCE FOR TOTAL METAL ANALYSES IN MEDIUM CONCENTRATION SAMPLES

Parameter	No. of Obs.	No. of Calc.	Average % Recovery	Std. Dev. % Recovery	Range % Recovery
Total Metals					
Aluminum	1	1	96	-	-
Antimony	1	1	87	-	-
Arsenic	1	0	-	-	-
Barium	1	1	101	-	-
Beryllium	1	1	78	-	-
Cadmium	1	1	62 *	-	-
Calcium	1	1	97	-	-
Chromium	1	1	91	-	-
Cobalt	1	1	87	-	-
Copper	1	1	98	-	-
Iron	1	1	91	-	-
Lead	1	1	108	-	-
Magnesium	1	0	-	-	-
Manganese	1	1	85	-	-
Mercury	1	1	106	-	-
Nickel	1	1	91	-	-
Potassium	1	1	86	-	-
Selenium	1	1	20 *	-	-
Silver	1	1	72 *	-	-
Sodium	1	1	81	-	-
Thallium	1	1	74 *	-	-
Tin	1	1	64 *	-	-
Vanadium	1	1	95	-	-
Zinc	1	1	85	-	-

* - outside DQO
- - not applicable

TABLE 3-1c ACCURACY PERFORMANCE FOR DISSOLVED METAL ANALYSES IN LOW CONCENTRATION SAMPLES

Parameter	No. of Obs.	No. of Calc.	Average % Recovery	Std. Dev. % Recovery	Range % Recovery
Dissolved Metals					
Aluminum	2	2	88	9.0	79 - 97
Antimony	2	2	99	15.5	83 - 114
Arsenic	2	2	93	5.0	88 - 98
Barium	2	2	88	4.5	83 - 92
Beryllium	2	2	84	4.0	80 - 88
Cadmium	2	2	110	14.0	96 - 124
Calcium	2	2	93	1.0	92 - 94
Chromium	2	2	119	28.1	91 - 147
Cobalt	2	2	90	5.0	85 - 95
Copper	2	2	103	1.0	102 - 104
Iron	2	2	128 *	31.1	97 - 159
Lead	2	2	107	1.0	106 - 108
Magnesium	2	2	73 *	15.6	57 - 88
Manganese	2	2	106	4.5	101 - 110
Mercury	2	2	100	10.0	90 - 110
Nickel	2	2	101	19.0	82 - 120
Potassium	2	2	96	4.0	92 - 100
Selenium	2	2	80	3.0	77 - 83
Silver	2	2	91	13.0	78 - 104
Sodium	2	2	91	5.5	85 - 96
Thallium	2	2	83	6.0	77 - 89
Tin	2	2	76	9.0	67 - 85
Vanadium	2	2	95	5.0	90 - 100
Zinc	2	2	92	6.0	86 - 98

* - outside DQO
- - not applicable

TABLE 3-1d ACCURACY PERFORMANCE FOR DISSOLVED METAL ANALYSES IN MEDIUM CONCENTRATION SAMPLES

Parameter	No. of Obs.	No. of Calc.	Average % Recovery	Std. Dev. % Recovery	Range % Recovery
Dissolved Metals					
Aluminum	1	1	84	-	-
Antimony	1	1	37 *	-	-
Arsenic	1	1	84	-	-
Barium	1	1	96	-	-
Beryllium	1	1	76	-	-
Cadmium	1	1	68 *	-	-
Calcium	1	0	-	-	-
Chromium	1	1	80	-	-
Cobalt	1	1	76	-	-
Copper	1	1	53 *	-	-
Iron	1	1	76	-	-
Lead	1	1	0 *	-	-
Magnesium	1	1	81	-	-
Manganese	1	1	88	-	-
Mercury	1	1	60 *	-	-
Nickel	1	1	92	-	-
Potassium	1	0	-	-	-
Selenium	1	1	90	-	-
Silver	1	1	62 *	-	-
Sodium	1	0	-	-	-
Thallium	1	1	0 *	-	-
Tin	1	1	34 *	-	-
Vanadium	1	1	90	-	-
Zinc	1	1	92	-	-

* - outside DQO
- - not applicable

TABLE 3-2a MATRIX SPIKE RECOVERIES OUTSIDE OF PROGRAM DQO LIMITS FOR TOTAL METALS IN LOW CONCENTRATION SAMPLES

Parameter	MQB312	MQB319	MQB321
Total Metals			
Aluminum	-	NR	
Antimony	-	-	
Arsenic	T	T	
Barium	-	-	
Beryllium	-	-	
Cadmium	-	T	
Calcium	-	-	
Chromium	-	-	
Cobalt	-	-	
Copper	-	-	
Iron	-	NR	
Lead	-	T	
Magnesium	T	-	
Manganese	-	-	
Mercury	-		
Nickel	-	-	
Potassium	-	-	
Selenium	T	T	
Silver	-	-	
Sodium	-	-	
Thallium	-	-	
Tin	T	-	
Vanadium	-	-	
Zinc	-	-	

T - Total Metal, outside DQO.
- - Spike recovery within DQO.
NR - Not calculated (sample result > 4x spike)

TABLE 3-2b MATRIX SPIKE RECOVERIES OUTSIDE OF PROGRAM DQO LIMITS FOR TOTAL METALS IN MEDIUM CONCENTRATION SAMPLES

Parameter	MQB326
Total Metals	
Aluminum	-
Antimony	-
Arsenic	NR
Barium	-
Beryllium	-
Cadmium	T
Calcium	-
Chromium	-
Cobalt	-
Copper	-
Iron	-
Lead	-
Magnesium	NR
Manganese	-
Mercury	-
Nickel	-
Potassium	-
Selenium	T
Silver	T
Sodium	-
Thallium	T
Tin	T
Vanadium	-
Zinc	-

T - Total Metal, outside DQO.
- - Spike recovery within DQO.
NR - Not calculated (sample result > 4x spike)

TABLE 3-2c MATRIX SPIKE RECOVERIES OUTSIDE OF PROGRAM DQO LIMITS FOR DISSOLVED METALS IN LOW CONCENTRATION SAMPLES

Parameter	MQB312	MQB319	MQB321
Dissolved Metals			
Aluminum	-	-	
Antimony	-	-	
Arsenic	-	-	
Barium	-	-	
Beryllium	-	-	
Cadmium	-	-	
Calcium	-	-	
Chromium	-	D	
Cobalt	-	-	
Copper	-	-	
Iron	-	D	
Lead	-	-	
Magnesium	-	D	
Manganese	-	-	
Mercury	-		-
Nickel	-	-	
Potassium	-	-	
Selenium	-	-	
Silver	-	-	
Sodium	-	-	
Thallium	-	-	
Tin	D	-	
Vanadium	-	-	
Zinc	-	-	

D - Dissolved Metal, outside DQO.
- - Spike recovery within DQO.
NR - Not calculated (sample result > 4x spike)

TABLE 3-2d MATRIX SPIKE RECOVERIES OUTSIDE OF PROGRAM DQO LIMITS FOR DISSOLVED METALS IN MEDIUM CONCENTRATION SAMPLES

Parameter	MQB306	MQB326
Dissolved Metals		
Aluminum	-	
Antimony	D	
Arsenic	-	
Barium	-	
Beryllium	-	
Cadmium	D	
Calcium	NR	
Chromium	-	
Cobalt	-	
Copper	D	
Iron	-	
Lead	D	
Magnesium	-	
Manganese	-	
Mercury		D
Nickel	-	
Potassium	NR	
Selenium	-	
Silver	D	
Sodium	NR	
Thallium	D	
Tin	D	
Vanadium	-	
Zinc	-	

D - Dissolved Metal, outside DQO.
- - Spike recovery within DQO.
NR - Not calculated (sample result > 4x spike)

TABLE 3-3a PRECISION PERFORMANCE FOR TOTAL METAL ANALYSES IN LOW CONCENTRATION SAMPLES

Parameter	No. of Obs.	No. of Calc.	Average RPD	Std. Dev. RPD	Range RPD
Total Metals					
Aluminum	2	1	46.0 *	-	-
Antimony	2	0	-	-	-
Arsenic	2	0	-	-	-
Barium	2	0	-	-	-
Beryllium	2	0	-	-	-
Cadmium	2	0	-	-	-
Calcium	2	2	1.0	0.0	-
Chromium	2	1	3.0	-	-
Cobalt	2	0	-	-	-
Copper	2	1	13.0	-	-
Iron	2	2	7.5	6.4	3.0 - 12.0
Lead	2	0	-	-	-
Magnesium	2	2	2.0	1.4	1.0 - 3.0
Manganese	2	2	2.5	3.5	0.0 - 5.0
Mercury	2	0	-	-	-
Nickel	2	1	1.0	-	-
Potassium	2	1	6.0	-	-
Selenium	2	0	-	-	-
Silver	2	0	-	-	-
Sodium	2	2	2.5	0.7	2.0 - 3.0
Thallium	2	0	-	-	-
Tin	2	0	-	-	-
Vanadium	2	0	-	-	-
Zinc	2	0	-	-	-

* - outside DQO
- - not applicable

TABLE 3-3b PRECISION PERFORMANCE FOR TOTAL METAL ANALYSES IN MEDIUM CONCENTRATION SAMPLES

Parameter	No. of Obs.	No. of Calc.	Average RPD	Std. Dev. RPD	Range RPD
Total Metals					
Aluminum	1	1	8.0	-	-
Antimony	1	0	-	-	-
Arsenic	1	1	18.0	-	-
Barium	1	0	-	-	-
Beryllium	1	0	-	-	-
Cadmium	1	0	-	-	-
Calcium	1	1	1.0	-	-
Chromium	1	0	-	-	-
Cobalt	1	0	-	-	-
Copper	1	1	6.0	-	-
Iron	1	1	1.0	-	-
Lead	1	0	-	-	-
Magnesium	1	1	4.0	-	-
Manganese	1	1	2.0	-	-
Mercury	1	0	-	-	-
Nickel	1	1	14.0	-	-
Potassium	1	1	1.0	-	-
Selenium	1	1	65.0 *	-	-
Silver	1	0	-	-	-
Sodium	1	1	2.0	-	-
Thallium	1	0	-	-	-
Tin	1	0	-	-	-
Vanadium	1	0	-	-	-
Zinc	1	1	16.0	-	-

* - outside DQO
- - not applicable

TABLE 3-3c PRECISION PERFORMANCE FOR DISSOLVED METAL ANALYSES IN LOW CONCENTRATION SAMPLES

Parameter	No. of Obs.	No. of Calc.	Average RPD	Std. Dev. RPD	Range RPD
Dissolved Metals					
Aluminum	2	0	-	-	-
Antimony	2	0	-	-	-
Arsenic	2	0	-	-	-
Barium	2	0	-	-	-
Beryllium	2	0	-	-	-
Cadmium	2	1	20.0	-	-
Calcium	2	2	10.0	11.3	2.0 - 18.0
Chromium	2	0	-	-	-
Cobalt	2	0	-	-	-
Copper	2	0	-	-	-
Iron	2	1	3.0	-	-
Lead	2	1	5.0	-	-
Magnesium	2	2	2.5	0.7	2.0 - 3.0
Manganese	2	2	11.5	9.2	5.0 - 18.0
Mercury	2	0	-	-	-
Nickel	2	1	10.0	-	-
Potassium	2	1	0.0	-	-
Selenium	2	0	-	-	-
Silver	2	0	-	-	-
Sodium	2	2	8.5	7.8	3.0 - 14.0
Thallium	2	0	-	-	-
Tin	2	0	-	-	-
Vanadium	2	0	-	-	-
Zinc	2	0	-	-	-

* - outside DQO
- - not applicable

TABLE 3-3d PRECISION PERFORMANCE FOR DISSOLVED METAL ANALYSES IN MEDIUM CONCENTRATION SAMPLES

Parameter	No. of Obs.	No. of Calc.	Average RPD	Std. Dev. RPD	Range RPD
Dissolved Metals					
Aluminum	1	0	-	-	-
Antimony	1	0	-	-	-
Arsenic	1	1	19.0	-	-
Barium	1	1	3.0	-	-
Beryllium	1	1	10.0	-	-
Cadmium	1	0	-	-	-
Calcium	1	1	1.0	-	-
Chromium	1	0	-	-	-
Cobalt	1	1	4.0	-	-
Copper	1	1	14.0	-	-
Iron	1	1	0.0	-	-
Lead	1	0	-	-	-
Magnesium	1	0	-	-	-
Manganese	1	0	-	-	-
Mercury	1	0	-	-	-
Nickel	1	1	3.0	-	-
Potassium	1	1	0.0	-	-
Selenium	1	0	-	-	-
Silver	1	0	-	-	-
Sodium	1	1	1.0	-	-
Thallium	1	0	-	-	-
Tin	1	0	-	-	-
Vanadium	1	0	-	-	-
Zinc	1	0	-	-	-

* - outside DQO
- - not applicable

Table 3-4a ACCURACY PERFORMANCE FOR INORGANIC AND INDICATOR ANALYSES IN LOW CONCENTRATION SAMPLES

Parameter	No. of Obs.	No. of Calc.	Average % Recovery	Std. Dev. % Recovery	Range % Recovery
POC	2	2	86.5	4.5	82 - 91
POX	2	2	93	4.0	89 - 97
TOC	2	2	96	3.0	93 - 99
TOX	2	2	92	0.0	-
Total phenols	2	2	91.5	0.5	91 - 92
Nitrate nitrogen	2	2	100	0.0	-
Sulfate	2	2	115	25.1	90 - 140
Chloride	2	2	105	15.0	90 - 120
Cyanide	2	2	95	10.0	85 - 105
Bromide	2	2	95	5.0	90 - 100
Nitrite nitrogen	2	2	90	0.0	-
Fluoride	2	2	90	0.0	-
Sulfide	2	2	105	11.0	94 - 116

* - outside DQO
- - not applicable

Table 3-4b ACCURACY PERFORMANCE FOR INORGANIC AND INDICATOR ANALYSES IN MEDIUM CONCENTRATION SAMPLES

Parameter	No. of Obs.	No. of Calc.	Average % Recovery	Std. Dev. % Recovery	Range % Recovery
POC	1	1	111	-	-
POX	1	1	93	-	-
TOC	1	1	94	-	-
TOX	1	1	104	-	-
Total phenols	1	1	100	-	-
Nitrate nitrogen	1	1	100	-	-
Sulfate	1	1	70 *	-	-
Chloride	1	1	90	-	-
Cyanide	1	1	13 *	-	-
Bromide	1	1	100	-	-
Nitrite nitrogen	1	1	95	-	-
Fluoride	1	1	105	-	-
Sulfide	1	1	101	-	-

* - outside DQO
- - not applicable

Table 3-5a PRECISION PERFORMANCE FOR INORGANIC AND INDICATOR ANALYSES IN LOW CONCENTRATION SAMPLES

Parameter	No. of Obs.	No. of Calc.	Average RPD	Std. Dev. RPD	Range RPD
POC	2	2	6.5	2.1	5.0 - 8.0
POX	2	0	-	-	-
TOC	2	2	3.5	4.9	0.0 - 7.0
TOX	2	2	2.5	3.5	0.0 - 5.0
Total phenols	2	1	2.0	-	-
Nitrate nitrogen	2	0	-	-	-
Sulfate	2	2	0.0	0.0	-
Chloride	2	2	0.0	0.0	-
Cyanide	2	0	-	-	-
Bromide	2	0	-	-	-
Nitrite nitrogen	2	0	-	-	-
Fluoride	2	0	-	-	-
Sulfide	3	3	2.3	1.5	1.0 - 4.0

* - outside DQO
- - not applicable

Table 3-5b PRECISION PERFORMANCE FOR INORGANIC AND INDICATOR ANALYSES IN MEDIUM CONCENTRATION SAMPLES

Parameter	No. of Obs.	No. of Calc.	Average RPD	Std. Dev. RPD	Range RPD
POC	1	1	9.0	-	-
POX	1	1	10.0	-	-
TOC	1	1	2.0	-	-
TOX	1	1	1.0	-	-
Total phenols	1	0	-	-	-
Nitrate nitrogen	1	0	-	-	-
Sulfate	1	1	2.0	-	-
Chloride	1	1	0.0	-	-
Cyanide	1	1	14.0	-	-
Bromide	1	0	-	-	-
Nitrite nitrogen	1	0	-	-	-
Fluoride	1	0	-	-	-
Sulfide	1	1	8.0	-	-

* - outside DQO
- - not applicable

TABLE 3-6 ACCURACY PERFORMANCE FOR MATRIX SPIKE SAMPLES

Matrix Spike Compound	Pairs of Obs.	Average % Recovery	Std. Dev. % Recovery	Range % Recovery
Volatiles				
1,1-Dichloroethene	2	84.5	3.7	81 - 89
Trichloroethene	2	92.5	7.2	82 - 98
Chlorobenzene	2	94.8	6.9	89 - 104
Toluene	2	94.8	5.3	89 - 101
Benzene	2	101.5	6.5	93 - 107
Base/Neutrals				
1,2,4-Trichlorobenzene	3	72.0	5.3	65 - 79
Acenaphthene	3	59.3	5.8	55 - 69
2,4-Dinitrotoluene	3	65.3	10.9	55 - 81
Pyrene	3	69.7	5.3	63 - 76
N-Nitroso-di-n-propylamine	3	73.0	11.4	62 - 88
1,4-Dichlorobenzene	3	73.0	4.2	68 - 79
Acid Fraction				
Pentachlorophenol	3	37.8	50.4	4 - 111
Phenol	3	50.5	69.5	6 - 168
2-Chlorophenol	3	26.2 *	35.9	3 - 80
4-Chloro-3-methylphenol	3	53.2	34.8	25 - 99
4-Nitrophenol	3	15.0	18.7	3 - 42
Pesticides				
Lindane	3	77.0	22.7	59 - 107
Heptachlor	3	84.5	27.5	60 - 122
Aldrin	3	80.0	29.9	52 - 120
Dieldrin	3	85.2	8.5	77 - 96
Endrin	3	101.8	4.2	98 - 108
4,4'-DDT	3	94.0	10.9	82 - 108
Phos-Herbicide				
Phorate	3	107.6	9.2	100 - 122
Disulfoton	3	109.0	5.7	104 - 120
Parathion	3	123.3 *	12.2	109 - 139
Famphur	3	99.7	7.7	89 - 113
Chlor-Herbicide				
2,4-D	2	66.7	11.3	57 - 80
2,4,5-TP	2	85.3	13.0	69 - 98
2,4,5-T	2	82.3	7.0	72 - 88
Chlorobenzilate	2	61.3	19.6	44 - 82

(a) no range, both values are identical
* outside QQO
\- not applicable

Table 3-7a ACCURACY PERFORMANCE FOR SURROGATE SPIKE COMPOUNDS IN SAMPLES

Surrogate Spike Compound	Average % Recovery	Std Dev % Recovery	Range % Recovery
Volatiles			
Toluene-D8	100.1 (31) a	4.4	89 - 108
4-Bromofluorobenzene (BFB)	102.9 (31)	6.7	89 - 112
1,2-Dichloroethane-D4	98.6 (31)	9.0	88 - 114
Base/Neutrals			
Nitrobenzene-D5	68.1 (29)	12.0	40 - 86
2-Fluorobiphenyl	45.0 (29)	10.3	27 - 70
Terphenyl-D14	68.5 (29)	9.1	46 - 89
Acid Fraction			
Phenol-D5	45.3 (29)	25.3	2 - 81
2-Fluorophenol	38.9 (29)	23.0	1 - 66
2,4,6-Tribromophenol	35.5 (29)	17.6	3 - 63
Pesticide			
Dibutylchlorendate	80.1 (26)	10.1	58 - 99
Herbicide			
2,4-DB	98.5 (23)	24.4	54 - 185

a analyses per compound in parentheses.
* outside DQO
-- not applicable

Table 3-7b ACCURACY PERFORMANCE FOR SURROGATE SPIKE COMPOUNDS
IN MATRIX SPIKE/MATRIX SPIKE DUPLICATE SAMPLES

Surrogate Spike Compound	Average % Recovery	Std Dev % Recovery	Range % Recovery
Volatiles			
Toluene-D8	98.3 (4) a	1.9	97 - 101
4-Bromofluorobenzene (BFB)	99.8 (4)	2.2	98 - 103
1,2-Dichloroethane-D4	93.8 (4)	1.7	92 - 96
Base/Neutrals			
Nitrobenzene-D5	79.5 (6)	5.8	72 - 89
2-Fluorobiphenyl	50.7 (6)	3.2	47 - 56
Terphenyl-D14	65.5 (6)	10.1	53 - 77
Acid Fraction			
Phenol-D5	28.5 (6)	31.7	6 - 79
2-Fluorophenol	20.2 (6)*	29.8	1 - 63
2,4,6-Tribromophenol	20.3 (6)	28.4	1 - 59
Pesticide			
Dibutylchlorendate	87.5 (6)	5.2	81 - 95
Herbicide			
2,4-DB	78.0 (4)	12.8	66 - 91

a analyses per compound in parentheses.
* outside DQO
-- not applicable

Table 3-7c ACCURACY PERFORMANCE FOR SURROGATE SPIKE COMPOUNDS IN THE SAMPLING BLANKS

Surrogate Spike Compound	Average % Recovery	Std Dev % Recovery	Range % Recovery
Volatiles			
Toluene-D8	100.5 (2) a	0.7	100 - 101
4-Bromofluorobenzene (BFB)	108.5 (2)	0.7	108 - 109
1,2-Dichloroethane-D4	89.0 (2)	1.4	88 - 90
Base/Neutrals			
Nitrobenzene-D5	66.0 (2)	5.7	62 - 70
2-Fluorobiphenyl	42.0 (2)*	0.0	42 - 42
Terphenyl-D14	67.5 (2)	3.5	65 - 70
Acid Fraction			
Phenol-D5	68.5 (2)	7.8	63 - 74
2-Fluorophenol	59.0 (2)	5.7	55 - 63
2,4,6-Tribromophenol	43.5 (2)	2.1	42 - 45
Pesticide			
Dibutylchlorendate	85.5 (2)	3.5	83 - 88
Herbicide			
2,4-DB	97.5 (2)	7.8	92 - 103

a analyses per compound in parentheses.
* outside DQO
-- not applicable

TABLE 3-8 PRECISION PERFORMANCE FOR MATRIX SPIKE COMPOUNDS IN THE SAMPLES

Matrix Spike Compound	Pairs of Obs.	Average RPD	Std. Dev. RPD	Range RPD
Volatiles				
1,1-Dichloroethene	2	7.1	1.6	6.0 - 8.2
Trichloroethene	2	9.9	8.2	4.2 - 15.7
Chlorobenzene	2	11.0	6.4	6.5 - 15.5
Toluene	2	9.0	5.2	5.3 - 12.6
Benzene	2	9.9	4.5	6.8 - 13.1
Base/Neutrals				
1,2,4-Trichlorobenzene	3	2.8	1.6	1.4 - 4.5
Acenaphthene	3	4.3	2.9	1.8 - 7.5
2,4-Dinitrotoluene	3	7.4	4.0	5.1 - 12.0
Pyrene	3	5.8	3.8	2.7 - 10.1
N-Nitroso-di-n-propylamine	3	4.0	4.6	1.1 - 9.2
1,4-Dichlorobenzene	3	2.7	1.2	1.5 - 3.9
Acid Fraction				
Pentachlorophenol	3	18.9	20.1	0.0 - 40.0
Phenol	3	41.6	11.4	28.6 - 50.0
2-Chlorophenol	3	16.9	15.0	0.0 - 28.6
4-Chloro-3-methylphenol	3	21.9	19.1	3.1 - 41.2
4-Nitrophenol	3	5.1	8.9	0.0 - 15.4
Pesticides				
Lindane	3	8.6	5.1	3.3 - 12.7
Heptachlor	3	10.4	6.3	4.2 - 16.8
Aldrin	3	9.0	7.5	3.4 - 17.5
Dieldrin	3	4.5	4.7	1.0 - 9.9
Endrin	3	6.2	3.6	2.0 - 8.7
4,4'-DDT	3	6.7	6.2	1.9 - 13.6
Phos-Herbicide				
Phorate	3	4.4	5.3	0.0 - 10.3
Disulfoton	3	4.2	4.0	0.9 - 8.7
Parathion	3	9.5	5.2	3.7 - 13.7
Famphur	3	9.3	5.5	3.0 - 13.2
Chlor-Herbicide				
2,4-D	2	6.3	5.3	2.5 - 10.0
2,4,5-TP	2	10.6	7.6	5.2 - 16.0
2,4,5-T	2	10.6	13.3	1.2 - 20.0
Chlorobenzilate	2	6.3	5.7	2.2 - 10.3

a no range, both values are identical
* outside QQQ
\- not applicable

Table 3-9 PRECISION PERFORMANCE FOR SURROGATE COMPOUNDS
IN MATRIX SPIKE/MATRIX DUPLICATE SAMPLES

Surrogate Compound	Pairs of Obs	Average RPD	Std Dev RPD	Range RPD
Volatiles				
Toluene-D8	2	1.5	2.1	0.0 - 3.0
4-Bromofluorobenzene (BFB)	2	2.5	2.1	1.0 - 4.0
1,2-Dichloroethane-D4	2	1.6	0.7	1.1 - 2.1
Base/Neutrals				
Nitrobenzene-D5	3	7.0	4.3	3.7 - 11.9
2-Fluorobiphenyl	3	3.8	3.1	2.0 - 7.4
Terphenyl-D14	3	18.3	10.7	8.1 - 29.5
Acid Fraction				
Phenol-D5	3	33.1	6.1	28.6 - 40.0
2-Fluorophenol	3	5.1	8.9	0.0 - 15.4
2,4,6-Tribromophenol	3	35.7	55.8	0.0 -100.0
Pesticide				
Dibutylchlorendate	3	7.9	7.1	2.4 - 15.9
Herbicide				
2,4-DB	2	3.7	1.1	3.0 - 4.5

* outside DQO
-- not applicable

TABLE 3-10 ACCURACY PERFORMANCE FOR DIOXIN/FURAN SPIKE COMPOUNDS

Sample	== % Recovery of == 2,3,7,8-TCDD	1,2,3,4,7,8-HxCDD	1,2,3,7,8-PeCDF
BS CC#126372	107	99.8	101
BS CC#127763	112	111	101
MQB307N	109	88.2	103
MQB307ND	110	107	99.4

BS = Blank Spike
N = Native Spike
D = Laboratory Duplicate

4.0 CONCLUSIONS

4.1 Metals

The average percent recoveries for the metal analyses are within the program accuracy DQOs with the exceptions of Cd, Se for the low water total metals Cd, Se, Ag, Tl, Sn for total metal medium water, Fe, Mg for the low water dissolved metals. The average RPDs are within the program precision DQOs with the exceptions of Al for the low water total metals and Se for the medium water dissolved metals. * and Sb, Cd, Cu, Pb, Hg, Ag, Tl, Sn for the medium water dissolved metals.

Laboratory blank contamination was not reported but Na(T), Al(D), Pb(D), and Na(D) contamination was found in the sampling blanks. All requested analyses were completed.

For data usability evaluation, see the Inorganic Data Usability Audit Report. All evaluation criteria used for the metals analyses are based on IFB WA 84-J092 (SOW 785).

4.2 Inorganic and Indicator Parameters

The average percent recoveries for the inorganic and indicator analysis are within program accuracy DQOs with the exception of medium water $SO_4^=$ and CN^-. The average RPDs are within the program precision DQOs.

Laboratory blank contamination was not reported but $SO_4^=$, S^-, POC and TOX contamination was found in the sampling blanks. All requested analyses were completed.

For data usability evaluation, see the Inorganic Data Usability Audit Report. All evaluation criteria used for CN analysis are based on IFB WA 84-J092 (SOW 785).

4.3 Organics

4.3.1 Volatiles by purge and trap data quality

The data should be considered Quantitative for samples MQB309, MQB314, MQB315, MQB317, and MQB324. The remaining volatile samples should be considered suspect due to excessive holding time prior to analysis.

For data usability evaluation, see the Organic Data Usability Audit Report.

4.3.2 Semivolatile Data Quality

The semi-volatile analysis should be considered semi-quantitative on the condition that all samples were preserved and stored contractually. Holding time violations, QC spike recoveries and variability in response fator should be individually considered.

4.3.3 Pesticide Data Quality

The pesticide analyses should be considered qualitative with the exception of analytes whose individual standard did not meet qualitative criteria. The forementioned compounds should be considered suspect.

For data usability evaluation, see the Organic Data Usability Audit Report.

4.3.4 Herbicide Data Quality

The phosphoherbiide data should be considered qualitative and there is a possibility of false negaties in samples MQB306 and MQB326. The chloroherbicide data should be considered suspect.

For data usability evaluation, see the Organic Data Usability Audit Report.

4.4 Dioxin/Furan Data Quality

The recoveries of the dioxin/furan analytes from the native spiked blanks and samples range from 88-112%. RPDs are incalculable because target analytes were not detected in the duplicate field and laboratory samples.

Th Dioxin/Furan analyes is considered semiquantitative with elevated detection limits.

For data usability evaluation, see the Dioxin/Furan Data Usability Audit Report.

APPENDIX 1

Contract Required Detection Limits and Instrument Detection Limits for Metals, Inorganic, and Indicator Parameters

TABLE A1-1

CONTRACT REQUIRED DETECTION LIMITS AND INSTRUMENT DETECTION LIMITS FOR METALS, INORGANIC, AND INDICATOR PARAMETERS

Parameter	CRDL	IDL
Metals		
Aluminum	200	94
Antimony	60	5
Arsenic	10	6
Barium	200	3
Beryllium	5	2
Cadmium	5	0.5
Calcium	5000	67
Chromium	10	6
Cobalt	50	7
Copper	25	18
Iron	100	23
Lead	5	2
Magnesium	5000	84
Manganese	15	4
Mercury	0.2	0.2
Nickel	40	23
Potassium	5000	486
Selenium	5	4
Silver	10	5
Sodium	5000	163
Tin	50	72
Thallium	10	6
Vanadium	50	8
Zinc	20	20
Inorganic and Indicators		
Bromide	1000	50
Chloride	1000	1000
Cyanide	10	
Fluoride	1000	1000
Nitrate-nitrogen	300	300
Nitrite-nitrogen	300	50
POC	100	20
POX	5	5
Sulfate	1000	500
Sulfide	1000	1000
TOC	1000	1000
TOX	5	5
Total Phenols	50	10

concentrations are in µg/l

APPENDIX 2

SUMMARY OF CONCENTRATIONS FOR COMPOUNDS FOUND IN GROUND-WATER AND SAMPLING BLANK SAMPLES AT CWM, VICKORY, OH

The following table lists the concentrations for compounds analyzed for and found in samples at the site. Table A2-1 is generated by listing all compounds detected and all tentatively identified compounds reported on the organic Form I, Part B. All tentatively identified compounds with a spectral purity greater than 850 are identified by name and purity in the table. Those with a purity of less than 850 are labeled, unknown.

TABLE KEY

A value without a flag indicates a result above the contract required detection limit (CRDL).

J Indicates an estimated value. This flag is used either when estimating a concentration for tentatively identified compounds where a 1:1 response is assumed or when the mass spectral data indicated the presence of a compound that meets the identification criteria but the result is less than the specified detection limit but greater than zero. If the limit of detection is 10 µg and a concentration of 3 µg is calculated, then report as 3J.

B This flag is used when the analyte is found in the blank as well as a sample. It indicates possible/probable blank contamination and warns the data user to take appropriate action.

GW = ground-water
SW = surface-water
low and medium are indicators of concentration.

Results for the samples reanalyzed and/or reextracted are preceeded by a / (slash).

All concentrations are in µg/L.

SITE: CHEM WASTE
CASE NO:10242/M

SAMPLE NO:		M0R301	M0R304	M0R311	M0R314	M0R315/ M0R315RA	M0R307	M0R310	M0R302	M0R307/ M0R307RE	M0R305	M0R316
SAMPLE LOCATION:		TRIP BLANK	FIELD BLANK	FIELD BLANK	WELL L-36 DUP	WELL L-36 DUP	WELL L-34 DUP	WELL L-34 DUP	WELL L-35	WELL L-15	WELL MW-219	WELL L
SAMPLE TYPE:		QW-LOW	QW-LOW	QW-LOW	SW-LOW	QW-LOW	QW-LOW	QW-LOW	QW-LOW	QW-LOW	QW-LOW	SW-MED
VOA	ACETONE	8 J B			3 JB	4 JB/7 JB				3 J		14000 B
	BENZENE											
	2-BUTANONE											
	CARBON DISULFIDE											
	CHLOROBENZENE											
	CHLOROFORM											2600
	1,1-DICHLOROETHANE											
	1,2-DICHLOROETHANE				1 J	1 1/2 J						
	2-HEXANONE											
	METHYLENE CHLORIDE				3 J					3 J		
	4-METHYL-2-PENTANONE											540 B
	TOLUENE											1600
	TRICHLOROETHANE											520
	TRICHLOROETHENE											150 J
ADD	1,4-DIOXANE											
VOA	ISOBUTYL ALCOHOL											4000 J
TIC-	ACETIC ACID, ETHYL ESTER											27000
VOA	BUTANE										PUR 929 4 J	
	CYCLOHEXANE											
	CYCLOPENTANE											
	CYCLOPENTANE, METHYL											
	METHANE, THIOBIS											
	3-PENTANONE											
	UNKNOWN ALIPHATIC ALCOHOL											PUR 966 1000J
	UNKNOWN											
	UNKNOWN											
SEMI-	BENZOIC ACID					7 J						190000
VOA	BIS(2-ETHYLHEXYL)PHTHALATE	5 J	3 J		3 J	9 J	3 J	5 J	4 J	3 J/17 B		
	2-CHLOROPHENOL											4200 J
	DI-N-BUTYLPHTHALATE				12	9 J		2 J				
	2,4-DIMETHYLPHENOL											5300 J
	4-METHYLPHENOL											25000
	PHENOL				3 J			2 J	3 J			330000
ADD	4-METHYL-2-PENTANONE				3 J							
SEMI	N-NITROSODIMORPHOLINE											
VOA												
SEMI-	1,3-PROPANEDIOL,2,2-DIMETHYL											
VOA TIC	2-CYCLOHEXEN-1-ONE					PUR 935 10 J						
	2-METHYLCYCLOPENTANOL ISOMERS							PUR 890 30 J		PUR 881 10 J		
	2-PROPANOL,1-(DIMETHYLAMINO)-2-METHYL				PUR 914 10 J	PUR 888 40 J						
	2-PROPENAMIDE,N-(1,1-DIMETHYL-ETHYL)									PUR 930 20		
	2-PROPENAMIDE,N-(1,1-DIMETHYL-3-OXOBUTYL)									PUR 926 /20 J		
										PUR 919 /200J		

SITE: OMEN WASTE
CASE #012743/M

SAMPLE NO:	M0R301	M0R304	M0R311	M0R314	M0R315/ M0R316RA	M0R307	M0R319	M0R302	M0R307/ M0R307RE	M0R305	M0R306
SAMPLE LOCATION:	TRIP BLANK	FIELD BLANK	FIELD BLANK	WELL L-26 DUP	WELL L-26 DUP	WELL L-34 DUP	WELL L-34 DUP	WELL L-35	WELL L-15	WELL MW-21D	WELL L
SAMPLE TYPE:	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-MED
3-CYCLOHEXENE-1- CARBOXYLIC ACID											PUR 827 40000 J
ACETIC ACID, PHENOXY											PUR828 100000 J
BENZALDEHYDE, 4-HYDROXY											PUR 915 20000 J
BENZENE, 1-CHLORO-2-ETHENYL											PUR899 100000 J
BICYCLO (2.2.1) HEPT-5-ENE- 2,3-DICARBOXYLIC ACID				PUR 717 30 J	PUR 751 100 J						
1,4,5,6,7,7-HEXACHLORO											
BUTANE											
CYCLOHEXANE											
CYCLOPENTANE											
CYCLOPENTANE,METHYL											
DIACETONE ACRYLAMIDE						PUR 904 20 J	PUR 922 10 J		PUR 919 300 J		
HEXADECANOIC ACID				PUR 741 9 J							
HEXANOIC ACID, 2-ETHYL											PUR 909 20000 J
O-CHLOROANILINE											
PHENOL, 4,4-METHYLENEBIS											PUR815 40000 J
SULFAR,MOL (S8)											
UNKNOWN	100 J	70 J	40 J	100 J	20 J	40 J	50 J	9 J	10 J/8 J	50 J	10000 J
UNKNOWN	30 J	10 J		7 J	20 J	8 J	20 J	40 J	10 J/10 J		40000 J
UNKNOWN	20 J			7 J	20 J			20 J	30 J/30 J		
UNKNOWN				10 J	100 J				9 J/70 J		
UNKNOWN				10 J	20 J				80 J/20 J		
UNKNOWN				40 JB					5 J/20 JB		
UNKNOWN				9 J					7 J/200 JB		
UNKNOWN				30 J					20 J/60 J		
UNKNOWN				40 JB					6 J		
UNKNOWN									10 J		
UNKNOWN									100 J		
UNKNOWN									20 J		
UNKNOWN											
UNKNOWN											
UNKNOWN											
UNKNOWN ALICYCLIC HYDROCARBON					20 J						
UNKNOWN ALICYCLIC SULFUR COMPOUND				10 J							
UNKNOWN ALICYCLIC SULFUR COMPOUND				10 J							
UNKNOWN ALICYCLIC SULFUR COMPOUND				10 J							
UNKNOWN ALICYCLIC SULFUR COMPOUND											
UNKNOWN ALICYCLIC SULFUR COMPOUND											
UNKNOWN ALICYCLIC SULFUR COMPOUND											
UNKNOWN ALICYCLIC SULFUR COMPOUND											
UNKNOWN ALICYCLIC											

SITE: CHEM WASTE
CASE NO:12343/M

SAMPLE NO:	MW301	MW304	MW311	MW314	MW314/ MW314DP	MW307	MW319	MW302	MW303/ MW303DP	MW305	MW306
SAMPLE LOCATION:	TRIP BLANK	FIELD BLANK	FIELD BLANK	WELL L-24 DUP	WELL L-24 DUP	WELL L-24 DUP	WELL L-24 DUP	WELL L-25	WELL L-15	WELL MW-219	WELL L
SAMPLE TYPE:	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-MED
SULFUR COMPOUND											
UNKNOWN ALICYCLIC											
SULFUR COMPOUND											
UNKNOWN ALIPHATIC											
SULFUR COMPOUND											
UNKNOWN ALIPHATIC											
SULFUR COMPOUND											
UNKNOWN ALIPHATIC											
SULFUR COMPOUND											
UNKNOWN AROMATIC											
SULFUR COMPOUND											
UNKNOWN AROMATIC											
SULFUR COMPOUND											
UNKNOWN ALIPHATIC COMPOUND					20 J						
UNKNOWN ALIPHATIC COMPOUND					20 J						
UNKNOWN ALIPHATIC COMPOUND					40 J						
UNKNOWN ALIPHATIC COMPOUND					30 J						
UNKNOWN ALIPHATIC COMPOUND					20 J						
UNKNOWN ALIPHATIC											
NITROGEN COMPOUND											
UNKNOWN ALKANE				10 J	30 J						
UNKNOWN ALKENE				10 J	10 J						
UNKNOWN ALKYL ISOCYANATES									/10 J		
UNKNOWN ALKYLAMIDE	10 J	10 JB			10 JB		9 J		8 J/10 J	8 J	
UNKNOWN ALKYLAMIDE	10 J				20 J				10 J/8 J		
UNKNOWN ALKYLAMIDE									10 JB/70 J		
UNKNOWN ALKYLAMIDE									10 J		
UNKNOWN ALKYLAMIDE									30 J		
UNKNOWN ALKYLAMIDE									30 J		
UNKNOWN CARBOXYCYCLIC ACID					20 J						5000 J
UNKNOWN CARBOXYCYCLIC ACID					20 J					20 J	
UNKNOWN CYCLIC ALCOHOL											
UNKNOWN HYDROCARBON											30000 J
UNKNOWN MONOCHLORINATED AROMATIC											
UNKNOWN PHOSPHORODITHIOIC ACID ESTER											10000 J
UNKNOWN PHOSPHORODITHIOIC ACID ESTER											
UNKNOWN PHOSPOROTHIOIC ACID											
UNKNOWN PHOSPOROTHIOIC ACID											
UNKNOWN SUBSTITUTED PHENOL											
UNKNOWN SUBSTITUTED PHENOL											
UNKNOWN SULFUR COMPOUND											
UNKNOWN SULFUR COMPOUND											
UNKNOWN ALKYAMIDE									100 J		
X-CHLOROBENZOIC ACID											

SAMPLE NO:	MW304
SAMPLE LOCATION:	WELL L
SAMPLE TYPE:	GW-MED
UNKNOWN ALKYLAMIDE	5000 J
UNKNOWN CARBOXYCYCLIC ACID	
UNKNOWN CYCLIC ALCOHOL	
UNKNOWN HYDROCARBON	
UNKNOWN MONOCHLORINATED AROMATIC	30000 J
UNKNOWN PHOSPHORODITHIOIC ACID ESTER	10000 J
UNKNOWN PHOSPHORODITHIOIC ACID ESTER	
UNKNOWN PHOSPOROTHIOIC ACID	
UNKNOWN PHOSPOROTHIOIC ACID	
UNKNOWN SUBSTITUTED PHENOL	
UNKNOWN SUBSTITUTED PHENOL	
UNKNOWN SULFUR COMPOUND	
UNKNOWN SULFUR COMPOUND	
UNKNOWN ALKYAMIDE	
X-CHLOROBENZOIC ACID	PUR 850 10000 JJ

PEST/ NONE DETECTED

SITE: CHEM WASTE
CASE #012747/M

SAMPLE NO:		M09701	M09704	M09711	M09714	M09714/ M09714RA	M09707	M09719	M09702	M09703/ M09703RE	M09705	M09706
SAMPLE LOCATION:		TRIP BLANK	FIELD BLANK	FIELD BLANK	WELL L-34 DUP	WELL L-34 DUP	WELL L-34 DUP	WELL L-34 DUP	WELL L-33	WELL L-15	WELL MW-21R	WELL L
SAMPLE TYPE:		SW-LOW	SW-LOW	SW-LOW	SW-LOW	SW-LOW	SW-LOW	SW-LOW	SW-LOW	SW-LOW	SW-LOW	SW-MED
PCB												
VOLTD ORGS	2-4-5 T											
HERB												
PHOS HERB	NONE DETECTED											
PESTCH/ PURGN	NONE DETECTED											
TOTAL METALS	ALUMINUM	54			4700	4170	4900	8240	7270	1280	90	288
	ANTIMONY	3										33
	ARSENIC							9.2				12300
	BARIUM			13	70	69	90	105	76	50		567
	BERYLLIUM											
	CADMIUM						3			3.1		
	CALCIUM		195	541	309000	307000	253000	244000	152000	885000	433000	5890000
	CHROMIUM				40	46	392	390	57	16		
	COBALT											142
	COPPER				41	42	35	36	35	16		475
	IRON			25	4850	4670	11100	13400	13700	1760	292	1420
	LEAD				9.6	9.5	12	13	15	2.4		
	MAGNESIUM				108000	107000	102000	98600	76200	362000	190000	1440
	MANGANESE				16900	16900	401	416	181	991	11	
	MERCURY											
	NICKEL				119	119	194	206	113	285		1240
	POTASSIUM				3910	3610	4010	5180	4850	4280	13900	2560000
	SELENIUM											6.6
	SILVER											
	SODIUM	160000	289	847	478000	490000	33200	32100	32700	333000	462000	3650000
	THALLIUM											
	TIN											
	VANADIUM							15				
	ZINC			15	35	23	48	47	89	51		19
DIS METALS	ALUMINUM	172		259	154	128	74	155		215	136	287
	ANTIMONY											
	ARSENIC											20800
	BARIUM	8		7	36	35	46	47	42	42		562
	BERYLLIUM	2							5			10
	CADMIUM							1.8		2.8		4
	CALCIUM	196		527	276000	285000	218000	204000	151000	879000	627000	6400000
	CHROMIUM											

SITE: CHEM WASTE
CASE NO:12743/M

SAMPLE NO:		M09701	M09704	M09711	M09714	M09714/ M09715S	M09707	M09719	M09702	M09707/ M09703S	M09705	M09706
SAMPLE LOCATION:		TRIP BLANK	FIELD BLANK	FIELD BLANK	WELL L-26 DUP	WELL L-24 DUP	WELL L-34 DUP	WELL L-34 DUP	WELL L-25	WELL L-15	WELL MW-21R	WELL L
SAMPLE TYPE:		GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-MED
	COBALT											170
	COPPER				12	10			24	12		200
	IRON	77		26	55	55				21	206	1400
	LEAD		16	4.8		16		19			17	
	MAGNESIUM	460		456	102000	105000	94000	104000	78200	365000	204000	1940
	MANGANESE				19400	16800	395	277	23	890	11	
	MERCURY											
	NICKEL				93	161	99	112	37	230		1240
	POTASSIUM	1170		1090	2530	2450	2520	2750	4120	4450	17200	2720000
	SELENIUM											34
	SILVER											
	SODIUM	162000	177000	1120	736000	709000	31200	38500	35100	320000	1240000	4070000
	THALLIUM											
	TIN											
	VANADIUM											
	ZINC									14		14
										45		
INORG.	BROMIDE											100000
INDIC.	CHLORIDE				1000000	875000	218000	200000	14000	2330000	448000	10000000
	CYANIDE											4330
	FLUORIDE											640000
	NITRATE NITROGEN											120000
	NITRITE NITROGEN											
	POC	32	40	220	28	20	39	50	48	86	660	14200
	POX											2700
	SULFATE		1880000		550000	550000	220000	185000	275000	810000	1870000	1180000
	SULFIDE			217000	704000	674000	736000	700000	662000	346000		1440000
	TOC				49000	49000	5100	4600	1400	27000	3200	8500000
	TOTAL PHENOLS				59							613000
	TOX		9.4		2000	1760	18	18		162	15	382000

HCCP

SITE: CHEM WASTE
CASE #012763/M

	SAMPLE NO:	M09300	M09309/ M09309RA	M09310	M09312	M09313	M09315/ M09315RA	M09317/ M09317RA/RE	M09319	M09320/ M09320RA	M09321	M09322
	SAMPLE LOCATION:	WELL MW-11R	WELL MW-27R	WELL P-10	WELL MW-16R	MAYERS CREEK	WELL L-27	WELL L-30	WELL L-21	WELL L-29	WELL L-31	WELL L-39
	SAMPLE TYPE:	GW-LOW	GW-LOW	GW-LOW	GW-LOW	SW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW
VOA	ACETONE					1 J		3 J/6 J		3 J/6 J	3 J	
	BENZENE			12								
	2-BUTANONE					5 J						
	CARBON DISULFIDE											
	CHLOROBENZENE											
	CHLOROFORM		/1 J									
	1,1-DICHLOROETHANE											
	1,2-DICHLOROETHANE											
	2-HEXANONE											
	METHYLENE CHLORIDE	3 J		6 B		1 J	2 B/11 B	1 B	2 J	/1 B		
	4-METHYL-2-PENTANONE											
	TOLUENE			5								
	TRICHLOROETHANE											
	TRICHLOROETHENE											
ADD. VOA	1,4-DIOXANE											
	ISOBUTYL ALCOHOL											
TIC-VOA	ACETIC ACID, ETHYL ESTER											
	BUTANE			PUR 900 20 J								
	CYCLOHEXANE			PUR 927 3 J								
	CYCLOPENTANE			PUR 941 5 J								
	CYCLOPENTANE, METHYL			PUR 935 20 J								
	METHANE, THIOBIS											
	3-PENTANONE											
	UNKNOWN ALIPHATIC ALCOHOL											
	UNKNOWN			30 J								
	UNKNOWN			30 J								
SEMI-VOA	BENZOIC ACID											
	BIS(2-ETHYLHEXYL)PHTHALATE	3 J	5 J	4 J	2 J	4 J	3 J	3 J/66 B	3 J	3 J	2 J	2 J
	2-CHLOROPHENOL											
	DI-N-BUTYLPHTHALATE											
	2,4-DIMETHYLPHENOL											
	4-METHYLPHENOL											
	PHENOL											
ADD SEMI VOAS	4-METHYL-2-PENTANONE											
	N-NITROSOMORPHOLINE							4 J/9 J				
SEMI-VOA TIC	1,3-PROPANEDIOL,2,2-DIMETHYL							PUR 946 / 2 J				
	2-CYCLOHEXEN-1-ONE											
	2-METHYLCYCLOPENTANOL ISOMERS	PUR 904 50 J					PUR 881 8 J		PUR 898 10 J			
	2-PROPANOL,1-IODO-2-METHYL											
	2-PROPENAMIDE,N-(1,1-DIMETHYL-ETHYL)							PUR 942 /50 J				
	2-PROPENAMIDE,N-(1,1-DIMETHYL-3-OXOBUTYL)							PUR 933 100 J				

SITE: CHEM WASTE
CASE #012763/M

SAMPLE NO:	M0B309	M0B309/ M0B309DUP	M0B310	M0B312	M0B317	M0B315/ M0B315DUP	M0B317/ M0B317DUP/RE	M0B319	M0B320/ M0B320DUP	M0B321	M0B322
SAMPLE LOCATION:	WELL MW-14R	WELL MW-37R	WELL P-10	WELL MW-14R	HAYES CREEK	WELL L-27	WELL L-20	WELL L-21	WELL L-29	WELL L-31	WELL L-39
SAMPLE TYPE:	GW-LOW	GW-LOW	GW-MED	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW
3-CYCLOHEXENE-1-CARBOXYLIC ACID											
ACETIC ACID, PHENOXY											
BENZALDEHYDE, 4-HYDROXY											
BENZENE, 1-CHLORO-2-ETHENYL											
BICYCLO (2.2.1) HEPT-5-ENE-2,3-DICARBOXYLIC ACID 1,4,5,6,7,7-HEXACHLORO											
BUTANE											
CYCLOHEXANE											
CYCLOPENTANE											
CYCLOPENTANE,METHYL											
DIACETONE ACRYLAMIDE							PUR 917 300 J	PUR 919 50 J			
HEXADECANOIC ACID							PUR 645 4 J				
HEXANOIC ACID, 2-ETHYL											
O-CHLOROANILINE											
PHENOL, 4,4-METHYLENEBIS											
SULFAR,HOL (S8)			PUR 935 5000 J								
UNKNOWN	100 J	20 J	100 J	40 J	90 J	20 J	5 J/40 J	10 J	50 J	40 J	80 J
UNKNOWN	20 J	100 J	100 J		10 J	60 J	40 J/10 J	10 J			20 J
UNKNOWN		7 JB1	20 J				40 J/10 J	60 J			
UNKNOWN		20 J	30 J				30 J/10 J	10 J			
UNKNOWN			50 J				100 J/10 J				
UNKNOWN			8 J				10 J/10 J				
UNKNOWN							10 J/100 J				
UNKNOWN							8 J/30 J				
UNKNOWN							20 J/10 JB1				
UNKNOWN							5 J/200 JB1				
UNKNOWN							4 J/50 JB1				
UNKNOWN							90 J				
UNKNOWN							10 JB1				
UNKNOWN							5 J				
UNKNOWN											
UNKNOWN ALICYCLIC HYDROCARBON											
UNKNOWN ALICYCLIC SULFUR COMPOUND		500 J	500 J								
UNKNOWN ALICYCLIC SULFUR COMPOUND		200 J	10 J								
UNKNOWN ALICYCLIC SULFUR COMPOUND		300 J	200 J								
UNKNOWN ALICYCLIC SULFUR COMPOUND		200 J	100 J								
UNKNOWN ALICYCLIC SULFUR COMPOUND		20 J	10 J								
UNKNOWN ALICYCLIC SULFUR COMPOUND		300 J	100 J								
UNKNOWN ALICYCLIC SULFUR COMPOUND		50 J	40 J								
UNKNOWN ALICYCLIC											

SITE: CHEM WASTE
CASE #012743/M

SAMPLE NO:	M08309	M08309/ M08309DA	M08310	M08313	M08317	M08315/ M08315DA	M08317/ M08317DA/DE	M08319	M08320/ M08320DA	M08321	M08322
SAMPLE LOCATION:	WELL MW-14R	WELL MW-22R	WELL P-10	WELL MW-15R	MAYERS CREEK	WELL L-27	WELL L-30	WELL L-31	WELL L-29	WELL L-21	WELL L-29
SAMPLE TYPE:	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW
SULFUR COMPOUND		20 J	40 J								
UNKNOWN ALICYCLIC											
SULFUR COMPOUND		50 J									
UNKNOWN ALIPHATIC											
SULFUR COMPOUND		500 J									
UNKNOWN ALIPHATIC											
SULFUR COMPOUND		70 J									
UNKNOWN ALIPHATIC											
SULFUR COMPOUND		10 J									
UNKNOWN AROMATIC											
SULFUR COMPOUND		20 J									
UNKNOWN AROMATIC											
SULFUR COMPOUND		100 J									
UNKNOWN ALIPHATIC COMPOUND											
UNKNOWN ALIPHATIC COMPOUND											
UNKNOWN ALIPHATIC COMPOUND											
UNKNOWN ALIPHATIC COMPOUND											
UNKNOWN ALIPHATIC COMPOUND											
UNKNOWN ALIPHATIC											
NITROGEN COMPOUND							50RE J				
UNKNOWN ALKANE											
UNKNOWN ALKENE											
UNKNOWN ALKYL ISOCYANATES											
UNKNOWN ALKYLAMIDE	8 J	10 J			10 J	9 JR	2/10RE J	10 J		8 J	10 JR
UNKNOWN ALKYLAMIDE	10 JR	20 JR					90/70RE J	10 J			
UNKNOWN ALKYLAMIDE							20RE JR	8 J			
UNKNOWN ALKYLAMIDE							30RE JR	9 J			
UNKNOWN ALKYLAMIDE							10 J				
UNKNOWN ALKYLAMIDE											
UNKNOWN CARBOXYCYCLIC ACID											
UNKNOWN CARBOXYCYCLIC ACID											
UNKNOWN CYCLIC ALCOHOL				200 J							
UNKNOWN HYDROCARBON							2 J				
UNKNOWN MONOCHLORINATED AROMATIC											
UNKNOWN PHOSPHORODITHIOIC ACID ESTER											
UNKNOWN PHOSPHORODITHIOIC ACID ESTER											
UNKNOWN PHOSPOROTHIOIC ACID											
UNKNOWN PHOSPOROTHIOIC ACID											
UNKNOWN SUBSTITUTED PHENOL											
UNKNOWN SUBSTITUTED PHENOL											
UNKNOWN SULFUR COMPOUND		7 J	40 J								
UNKNOWN SULFUR COMPOUND			90 J								
UNKNOWN ALKYAMIDE											
X-CHLOROBENZOIC ACID											
PEST/ NONE DETECTED											

SITE: CHEM WASTE
CASE NO: 12343/M

SAMPLE NO:		M0B309	M0B309/ M0B309RA	M0B310	M0B312	M0B313	M0B315/ M0B315RA	M0B317/ M0B317RA/RE	M0B319	M0B320/ M0B320RA	M0B321	M0B322
SAMPLE LOCATION:		WELL MW-14R	WELL MW-33S	WELL P-10	WELL MW-14S	MAYERS CREEK	WELL L-27	WELL L-30	WELL L-21	WELL L-29	WELL L-31	WELL L-39
SAMPLE TYPE:		GW-LOW	GW-LOW	GW-MED	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW
SO2												
CYLISS 2-4-5 T HERB												
PHOS HERB	NONE DETECTED											
DIOXIN/ FURAN	NONE DETECTED											
TOTAL METALS	ALUMINUM	93	112	282	92	2050	3670	3510	3280	850	2730	3630
	ANTIMONY											
	ARSENIC		24.1									
	BARIUM		6	160		41	63	62	69	59	53	61
	BERYLLIUM											
	CADMIUM			0.5								
	CALCIUM	575000	1040000	1310000	610000	81700	139000	947000	599000	169000	154000	207000
	CHROMIUM						120	48	301	11	30	45
	COBALT											
	COPPER	10	9	9			34	40	44	15	19	30
	IRON	1390		329	659	2320	7440	6100	6720	1440	4540	4840
	LEAD						11		5		6.1	3.9
	MAGNESIUM	147000	350000	474000	158000	29500	55100	386000	363000	65400	134000	129000
	MANGANESE	19	14	9	20	35	479	3290	497	174	44	150
	MERCURY											
	NICKEL						102	80	332			44
	POTASSIUM	10400	54500	95340	12900	3440	3110	4710	2750	922	2350	2500
	SELENIUM											
	SILVER											
	SODIUM	56400	965000	1680000	64300	15300	18900	1010000	77700	14900	55000	39400
	THALLIUM											
	TIN											
	VANADIUM											
	ZINC						41	25	59		17	17
DIS METALS	ALUMINUM	185	188	231	115	235	179	143	169		117	62
	ANTIMONY											
	ARSENIC											
	BARIUM											
	BERYLLIUM			16		36	30	31	50	38	35	24
	CADMIUM			6		2			2		4	
	CALCIUM	553000	932000	1490000	594000	90000	114000	899000	0.7	156000	144000	168000
	CHROMIUM								575000			

SITE: CHEM WASTE
CASE NO:12343/M

SAMPLE NO:		M0B309	M0B309/ M0B309RA	M0B310	M0B312	M0B313	M0B315/ M0B315RA	M0B317/ M0B317RA/RE	M0B319	M0B320/ M0B320RA	M0B321	M0B322
SAMPLE LOCATION:		WELL MW-14R	WELL MW-23R	WELL P-10	WELL MW-16R	MAYERS CREEK	WELL L-27	WELL L-30	WELL L-21	WELL L-29	WELL L-31	WELL L-19
SAMPLE TYPE:		GW-LOW	GW-LOW	GW-MED	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW	GW-LOW
	COBALT											
	COPPER	12	9	35	1		11		15			
	IRON	1300			575	168	42	550	48		24	
	LEAD				9.7					12		51
	MAGNESIUM	134000	372000	503000	151000	33600	46300	420000	331000	84700	141000	119000
	MANGANESE	18	15	7	30	15	314	3400	426	179	15	6
	MERCURY											
	NICKEL							57	304			
	POTASSIUM	10200	2530	118000	12900	3640	2730	4690	3230	688	2290	1410
	SELENIUM											
	SILVER						9					
	SODIUM	51100	1050000	1990000	60000	16000	15000	1080000	73000	14400	57900	37400
	THALLIUM											
	TIN											
	VANADIUM			15								
	ZINC											
INORG.	BROMIDE											
INRIC.	CHLORIDE	56000	1400000	38000000	100000	28000	35000	2240000	710000	20000	18000	70000
	CYANIDE											
	FLUORIDE		340000	1400000				5600	4000			
	NITRATE NITROGEN		36000	1400000		2300		1100				9500
	NITRITE NITROGEN											
	POC	162	3400	8000	126	16	118	30	70	17	64	22
	POX		19200	18600								
	SULFATE	1820000	270000	2800000	1460000	104000	150000	1260000	755000	275000	420000	355000
	SULFIDE	62000	354000	780000		159000		52000		660000	676000	129000
	TOC	3400	1100	1200	2300	6400	1300	37000	12000	1200	2500	3500
	TOTAL PHENOLS											
	TOX	120	129	750	13	24	22	433	53	28		26

SITE: CHEM WASTE
CASE #01Z743/M

		MOB322/			
SAMPLE #0:		MOB323SE	MOB324	MOB325	MOB326
SAMPLE LOCATION:		WELL L-19	WELL T-19	WELL T-25	WELL L
SAMPLE TYPE:		GW-LOW	GW-LOW	GW-LOW	GW-MED
VOA	ACETONE		2 JB		220 B
	BENZENE				
	2-BUTANONE				
	CARBON DISULFIDE				2 J
	CHLOROBENZENE				7
	CHLOROFORM				1 J
	1,1-DICHLOROETHANE	2 J			
	1,2-DICHLOROETHANE	5			
	2-HEXANONE				
	METHYLENE CHLORIDE		2 JB		3 JB
	4-METHYL-2-PENTANONE				31
	TOLUENE				2 J
	TRICHLOROETHANE				
	TRICHLOROETHENE				
ADD. VOA	1,4-DIOXANE				
	ISOBUTYL ALCOHOL				450
TIC-VOA	ACETIC ACID, ETHYL ESTER				
	BUTANE				
	CYCLOHEXANE				
	CYCLOPENTANE				
	CYCLOPENTANE, METHYL				
	METHANE, THIOBIS				PUR 977 100 J
	3-PENTANONE				
	UNKNOWN ALIPHATIC ALCOHOL				5 J
	UNKNOWN				5 J
	UNKNOWN				
SEMI-VOA	BENZOIC ACID				15000
	BIS(2-ETHYLHEXYL)PHTHALATE	41 B	3 J	3 J	400 JB
	2-CHLOROPHENOL				
	DI-N-BUTYLPHTHALATE				
	2,4-DIMETHYLPHENOL				
	4-METHYLPHENOL				1200
	PHENOL				34000
ADD SEMI VOAS	4-METHYL-2-PENTANONE				
	N-NITROSOMORPHOLINE				
SEMI-VOA TIC	1,3-PROPANEDIOL,2,2-DIMETHYL				
	2-CYCLOHEXEN-1-ONE				
	2-METHYLCYCLOPENTANOL ISOMERS		PUR 900 200 J		
	2-PROPANOL,1-IODO-2-METHYL				
	2-PROPENAMIDE,N-	PUR 916 10 J			
	(1,1-DIMETHYL-ETHYL)	PUR 947 10 J			
	2-PROPENAMIDE,N-				
	(1,1-DIMETHYL-3-OXOBUTYL)	PUR 919 100J			

SITE: CHEM WASTE
CASE NO:12743/M

SAMPLE NO:	MOB321/ MOB322DE	MOB324	MOB325	MOB326
SAMPLE LOCATION:	WELL L-19	WELL T-19	WELL T-25	WELL L
SAMPLE TYPE:	GW-LOW	GW-LOW	GW-LOW	GW-MED
1-CYCLOHEXENE-1-CARBOXYLIC ACID				
ACETIC ACID, PHENOXY				PUR 925 5000 J
BENZALDEHYDE, 4-HYDROXY				
BENZENE, 1-CHLORO-3-ETHENYL				PUR871 10000 J
BICYCLO (2.2.1) HEPT-5-ENE-2,3-DICARBOXYLIC ACID 1,4,5,6,7,7-HEXACHLORO				
BUTANE				
CYCLOHEXANE				
CYCLOPENTANE				
CYCLOPENTANE,METHYL				
DIACETONE ACRYLAMIDE	PUR 921 /90 J			
HEXADECANOIC ACID				
HEXANOIC ACID, 2-ETHYL				
O-CHLOROANILINE				PUR767 10000 J
PHENOL, 4,4-METHYLENEBIS				
SULFUR,MOL (S8)				
UNKNOWN	10 J/9 J	40 J	20 J	5 J
UNKNOWN	10 J/10 J			900 J
UNKNOWN	10 J/20 J			5000 J
UNKNOWN	10 J/7 J			1000 J
UNKNOWN	60 J/40 J			1000 J
UNKNOWN	10 J/10 JB			900 J
UNKNOWN	10 J/200 JB			2000 JB
UNKNOWN	20 J/60 JB			
UNKNOWN				
UNKNOWN				
UNKNOWN				
UNKNOWN				
UNKNOWN				
UNKNOWN				
UNKNOWN ALICYCLIC HYDROCARBON				
UNKNOWN ALICYCLIC SULFUR COMPOUND				
UNKNOWN ALICYCLIC SULFUR COMPOUND				
UNKNOWN ALICYCLIC SULFUR COMPOUND				
UNKNOWN ALICYCLIC SULFUR COMPOUND				
UNKNOWN ALICYCLIC SULFUR COMPOUND				
UNKNOWN ALICYCLIC SULFUR COMPOUND				
UNKNOWN ALICYCLIC				

SITE: Chem WASTE
CASE NO:12363/M

SAMPLE NO:	MOR323/ MOR323RE	MOR324	MOR325	MOR326
SAMPLE LOCATION:	WELL L-19	WELL T-19	WELL T-25	WELL L
SAMPLE TYPE:	GW-LOW	GW-LOW	GW-LOW	GW-MED
SULFUR COMPOUND				
UNKNOWN ALICYCLIC				
SULFUR COMPOUND				
UNKNOWN ALIPHATIC				
SULFUR COMPOUND				
UNKNOWN ALIPHATIC				
SULFUR COMPOUND				
UNKNOWN ALIPHATIC				
SULFUR COMPOUND				
UNKNOWN AROMATIC				
SULFUR COMPOUND				
UNKNOWN AROMATIC				
SULFUR COMPOUND				
UNKNOWN ALIPHATIC COMPOUND				
UNKNOWN ALIPHATIC COMPOUND				
UNKNOWN ALIPHATIC COMPOUND				
UNKNOWN ALIPHATIC COMPOUND				
UNKNOWN ALIPHATIC COMPOUND				
UNKNOWN ALIPHATIC				
NITROGEN COMPOUND				
UNKNOWN ALKANE				
UNKNOWN ALKENE				
UNKNOWN ALKYL ISOCYANATES				
UNKNOWN ALKYLAMIDE	20 J/20 J			900 J
UNKNOWN ALKYLAMIDE	/30 JB			
UNKNOWN ALKYLAMIDE				
UNKNOWN ALKYLAMIDE				
UNKNOWN ALKYLAMIDE				
UNKNOWN ALKYLAMIDE				
UNKNOWN CARBOXYCYCLIC ACID				
UNKNOWN CARBOXYCYCLIC ACID				900 J
UNKNOWN CYCLIC ALCOHOL				
UNKNOWN HYDROCARBON				
UNKNOWN MONOCHLORINATED AROMATIC				
UNKNOWN PHOSPHORODITHIOIC ACID ESTER				2000 J
UNKNOWN PHOSPHORODITHIOIC ACID ESTER				20000 J
UNKNOWN PHOSPHOROTHIOIC ACID				1000 J
UNKNOWN PHOSPHOROTHIOIC ACID				
UNKNOWN SUBSTITUTED PHENOL				900 J
UNKNOWN SUBSTITUTED PHENOL				5000 J
UNKNOWN SULFUR COMPOUND				20000 J
UNKNOWN SULFUR COMPOUND				
UNKNOWN ALKYAMIDE				
Y-CHLOROBENZOIC ACID				
PEST/ NONE DETECTED				

SITE: CHEM WASTE
CASE NO:2363/M

		MOR327/ MOR327RE	MOR324	MOR325	MOR326
SAMPLE NO:					
SAMPLE LOCATION:		WELL T-19	WELL T-19	WELL T-25	WELL T
SAMPLE TYPE:		GW-LOW	GW-LOW	GW-LOW	GW-MED
PCB					
CHLORO HERB	2-4-5 T				940
PHOS HERB	NONE DETECTED				
DIOXIN/ FURAN	NONE DETECTED				
TOTAL METALS	ALUMINUM	1460	491	466	417
	ANTIMONY				29
	ARSENIC	7.1			1230
	BARIUM	106	52	14	84
	BERYLLIUM				
	CADMIUM			0.8	2
	CALCIUM	696000	22800	261000	623000
	CHROMIUM	12	22	13	10
	COBALT				22
	COPPER	17	14		49
	IRON	2320	1290	1160	1710
	LEAD				
	MAGNESIUM	227000	98800	151000	288000
	MANGANESE	2120	90	158	439
	MERCURY				
	NICKEL	175	291	71	103
	POTASSIUM	4220	7940	4340	150000
	SELENIUM				11
	SILVER				
	SODIUM	945000	47000	49900	514000
	THALLIUM				
	TIN				
	VANADIUM				
	ZINC	14		347	48
DIS METALS	ALUMINUM	185	74	178	277
	ANTIMONY				9.2
	ARSENIC				1430
	BARIUM	95	39	15	89
	BERYLLIUM			6	4
	CADMIUM	17			
	CALCIUM	649000	210000	295000	666000
	CHROMIUM				

SITE: CHEM WASTE
CASE NO:2363/M

	SAMPLE NO:	MOB323/ MOB323RE	MOB324	MOB325	MOB326
	SAMPLE LOCATION:	WELL L-19	WELL T-19	WELL T-25	WELL L
	SAMPLE TYPE:	GW-LOW	GW-LOW	GW-LOW	GW-MED
	COBALT				20
	COPPER	11			41
	IRON	212		97	1670
	LEAD	48	14	38	
	MAGNESIUM	218000	97000	174000	298000
	MANGANESE	2280	45	157	497
	MERCURY				
	NICKEL	177	201	50	134
	POTASSIUM	4430	3520	7440	233000
	SELENIUM				
	SILVER				
	SODIUM	1090000	43400	53000	653000
	THALLIUM				
	TIN				
	VANADIUM				
	ZINC	24		320	44
INORG.	BROMIDE				
INRIC.	CHLORIDE	775000	19000	16000	900000
	CYANIDE				125
	FLUORIDE				
	NITRATE NITROGEN	400			
	NITRITE NITROGEN				
	POC	290	25	76	114
	POX		14		22
	SULFATE	250000	233000	1050000	1000000
	SULFIDE		179000	514000	34000
	TOC	13000		1600	1000000
	TOTAL PHENOLS				81200
	TOX	189	15	650	41400

8/6/83

RISK ASSESSMENT FOR THE OLD HAZARDOUS WASTE DISPOSAL SITE AT VICKERY, OHIO

Prepared by

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August 16, 1983

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PREFACE

This report has been prepared for, and at the request of Bergson, Borkland, Margolis, and Adler (BBMA) as part of their internal investigation of operations and environmental contamination at the OLD hazardous waste disposal site operated by Chemical Waste Management, Inc. (CWM) in Vickery, Ohio. It is based on information supplied by BBMA, CWM and their technical contractors, including Environmental Testing and Certification Corporation (ETC), Environmental Research and Technology, Inc. (ERT), Golder Associates (Golder), and Weston, Inc. (Weston). As part of its standard practices, Clement Associates, Inc. (Clement) has attempted to verify the reliability of data supplied by BBMA, CWM and their contractors, including review of sampling designs, analytical procedures and quality assurance data included in the contractors' reports. In certain cases, Clement has suggested additional sampling or analytical work to fill gaps in data required for risk assessment. However, Clement did not take part in the design or execution of field studies, sample collection, or sample analyses. Thus, Clement makes no representation as to the completeness or accuracy of the data on which this report is based. Clement's responsibility is limited to the use of these data in preparing a reasonable scientific assessment of the risks that might arise during and after various remedial options.

Clement has specialized in the preparation of written assessments for toxic chemicals, and has conducted a number of risk assessments for permitted uncontrolled hazardous waste sites. For this purpose Clement has developed special techniques of risk assessment to deal with specific problems that arise at hazardous waste sites. These techniques are described briefly in Section II of this report.

SUMMARY

This report presents an assessment of possible risks that might result from the presence of PCBs and other chemicals at the OLD waste disposal site operated by Chemical Waste Management, Inc. (CWM) at Vickery, Ohio. It is based on chemical, hydrogeological, and other environmental data supplied by CWM and its technical consultants (see Appendix A), and upon toxicological data available to Clement Associates, Inc. (Clement). Assessment of risks is presented both for the present state of the site and for the conditions that would prevail under each of nine remedial options outlined by CWM in its presentation of July 28, 1983, to the Ohio EPA. Both long-term risks resulting from chemical exposure and short-term risks arising during remedial work are considered.

Section II of this report summarizes and discusses procedures used for risk assessment at hazardous waste sites. Risk assessment for toxic chemicals involves three separate steps: exposure assessment, toxicity assessment, and risk assessment. Each of these steps introduces a number of scientific uncertainties, even when extensive data on exposure and toxicity are available. In the case of hazardous waste sites, additional uncertainties are usually introduced by the multiplicity of chemicals present, multiple pathways of environmental transport and exposure, incomplete information about the nature and extent of chemical contamination, and the long time-scales for chemical

migration. For these reasons, risk assessments for hazardous waste sites are rarely accurate to better than an order of magnitude, and semiquantitative or verbal characterizations of the extent and magnitude of risk are often appropriate An approach is described which involves the identification of a limited number of chemicals of primary concern at each site and the exploration of a limited number of exposure pathways, using a "worst-case scenario" approach.

Section III of this report summarizes the toxicity of the chemicals of primary concern at the Vickery site. Primary attention is devoted to PCBs because of their presence at the site and their special regulatory status (Section IIIA). The toxicity of 14 other chemicals present in substantial quantities is summarized briefly, based on USEPA's Ambient Water Quality Criteria documents (Section IIIB). For each chemical, daily intakes are identified that correspond to very low carcinogenic risks and/or ample margins of safety for other types of risk (Figure 1 and Table 2). These risks are placed in the context of upper low-level risks experienced in the general population (Table 3).

Section IV of this report explores eight exposure scenarios at the Vickery site and assesses in semiquantitative terms the risks associated with each. With the probable exception of exposure to volatile organic compounds (other than PCBs) volatilizing from the lagoons, risks resulting from long-term exposure to materials migrating from the site are judged to

be negligible or very low for all remedial options, including the no-action option. The most important risks under most options are short-term risks arising during the remedial work itself. This report explores several of these risks, including chemical exposures and operational accidents to remedial personnel, on-site and off-site exposures to materials volatilizing during remedial excavation, and off-site traffic accidents. For all options involving substantial off-site transportation, traffic accidents are judged to be the most important single source of risk arising from the site.

Table A presents a summary characterization of the risks judged likely to arise under each of the remedial options. Risks resulting from chemical exposures and accidents arising during remedial work can probably be kept low by an effective safety plan, but cannot be eliminated altogether. However, these risks are similar under all remedial options and the bottom line of table A reflects only incremental risks associated with each option. Under option 1, 1A, 1B, and 5, these incremental risks are judged to be negligible or very low (i.e., there would be an adequate or greater margin-of-safety for all chemical exposures, and there would be no substantial off-site transportation). These options represent an improvement over the no-action options, for which there would probably be a less-than-adequate margin-of-safety resulting from exposure to organic chemicals (other than PCBs) volatilizing from the lagoon. Options 2, 3, 4B, 4A, and 6 would involve large-scale

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TABLE A

CHARACTERIZATION[1] OF RISKS ARISING UNDER VARIOUS REMEDIAL OPTIONS AT THE VICKERY, OHIO, SITE

Type of Risk	No Action	Remedial Option 1	1A	1B	2	3	4A	4B	5	6
Volatilization of PCBs from ponds	-	-	-	-	-	-	-	-	-	-
Surface water runoff of PCBs	-	-	-	-	-	-	-	-	-	-
Infiltration of PCBs into ground-water and transport off-site	0	0	-	-	-	-	-	-	-	-
Volatilization of other VOCs from ponds; on-site or off-site exposure	XX	0	-	-	-	-	-	-	-	-
Exposure of remedial workers to PCBs*	-	0	0	0	0	0	0	0	0	0
Release of other VOCs during remedial work; on-site or off-site exposure*	-	X	X	X	X	X	X	X	X	X
On-site accidents*	-	X	X	X	X	X	X	X	X	X
Transportation accidents	-	-	-	-	X	XX	XXX	XX	-	XXXX
Overall characterization	XX	0	-	-	X	XX	XXX	XX	-	XXXX

-, negligible (at least ample margin of safety);

0, very low (adequate margin of safety);

X, low (marginal margin of safety; 2-3 accidents expected);

XX, low-to-moderate (marginal to inadequate margin of safety; 6-7 accidents expected);

XXX, moderate (10-12 accidents expected; death possible);

XXXX, moderate-to-high (20-25 accidents expected; death likely).

*These risks are common to all remedial options and are expected to be minimized by a safety plan (see text); they are not included in the overall characterization, which classifies incremental risks associated with each option.

transportation of materials from the site for off-site disposal and would result in progressively increasing risk of injury, death, and chemical spills from off-site traffic accidents. At the extreme, option 6 would involve nearly 5 million miles of off-site transportation by heavy trucks and would statistically be expected to result in 20-25 traffic accidents, a number of injuries, and probably at least one death; this option is categorized as giving rise to moderate-to-high risks.

I. INTRODUCTION

This report presents a summary of the possible risks that might arise from the storage, treatment and disposal of certain chemicals at the OLD hazardous waste site operated by Chemical Waste Management, Inc. (CWM), at Vickery, Ohio. Primary attention is devoted to risks that might be posed by polychlorinated biphenyls (PCBs), which have recently been found as contaminants of oils, sludges, and other substances at the site. Emphasis is placed on PCBs because of their special regulatory status under the Toxic Substances Control Act, which has led to the design of specific remedial measures to contain, reduce, or eliminate the contamination of the environment with these materials. Some attention is devoted to PCDD which has recently been found in trace quantities at the site (see Appendix D). Limited attention is paid to risks that might be posed by other materials identified at the site, particularly volatile organic compounds that are found in association with PCBs and that would be affected by the remedial measures applied to PCBs. To the extent possible, this report assesses the risks associated with each of the nine remedial options proposed by CWM in its submission to the Ohio EPA dated 28 July, 1983, as well as with the situation that would arise if no further remedial actions were applied to the present contamination. The results of this risk assessment have already been presented in schematic summary form in Attachment B, Table 1, of CWM's submission

of 28 July 1983. This report provides documentation and background information for those summary characterizations of risk.

This report is in three main parts. The first is a brief description of scientific procedures available for conducting risk assessments at hazardous waste sites, and explains the limited precision attainable by such assessments. The second is a summary of toxicity data on PCBs and other contaminants identified at the OLD site. The third includes analyses of a number of scenarios for human exposure, and assesses the potential for exposure and risk arising under each scenario. This section utilizes technical information supplied by other contractors, including hydrogeological information developed by Golder Associates, chemical analyses of sludges and other materials by Environmental Testing and Certification Corporation, characterization of the site by CWM, and air monitoring by Environmental Resources and Technology, Inc. (see Appendix A for a list of primary information used). The report concludes with a summary and tabulation of results. Certain technical parts of the risk assessments are presented in Appendices B and C to this report.

II. METHODS FOR RISK ASSESSMENT AT HAZARDOUS WASTE SITES

A. General Methodology for Risk Assessment

Risk assessment for toxic chemicals follows a methodology which has now become fairly standardized. It involves three distinct steps: exposure assessment, hazard assessment, and risk assessment. In exposure assessment, data on the distribution of the chemical in the environment are assembled, and pathways leading to human exposure are identified. The human population at risk of exposure is enumerated, and the distribution and magnitude of exposures are calculated. The data are synthesized to yield a summary of the number and distribution of persons exposed to the chemical at different levels and frequencies. If sufficient data are available, the distribution of exposures may be formulated in statistical terms; more frequently, a limited number of high exposure groups may be identified.

In hazard assessment, information on the toxicity of the chemical is assessed by means of critical review of toxicological and epidemiological studies. The toxic effects of most concern at low exposure levels are identified, and numerical dose-response data are extracted from the studies under review. For toxic effects other than carcinogenesis, the lowest dose levels that cause adverse effects ("LOAELs") and the highest dose levels that cause no adverse effects ("NOAELs") are identified. For carcinogenesis, it is customary to assume that no absolute

thresholds (NOAELS) may exist, and the data are fitted to mathematical models of dose-response relationships to estimate the probability of response per unit dose ("unit risk") at low-dose levels.

In risk assessment, the exposure and hazard data are synthesized to estimate the extent and magnitude of risks to the human population. For effects other than carcinogenesis, "margins of safety" between expected exposure levels and NOAELs or LOAELs are calculated for different segments of the population. For carcinogenic effects, data on the distribution of exposures are combined with estimates of unit risk to yield estimates of the distribution of risk. A scientific risk assessment should include not only a description of the risks to the population but also a statement of the scientific uncertainties in this description.

Interpretation of the results of a risk assessment involves not only scientific judgments about the probability of adverse effects but policy judgments about the acceptability of low risks. As a scientific matter, estimates of risk at low exposure levels are subject to considerable uncertainty. For effects other than carcinogenesis, fairly large margins of safety are required before it can be concluded that population risks are low. This requirement for large margins of safety arises both because humans may be more sensitive to low doses of a chemical than the animal species used in toxicity studies, and because the human population is variable, so that some individuals

may be much more sensitive than others. However, the magnitude of the margin of safety required in any specific case is a matter for scientific judgment. Toxicologists usually require at least a 100-fold margin of safety between a NOAEL established in an animal species and human exposure levels, and in some circumstances a 1000-fold margin of safety may be required before it can be stated with scientific confidence that risks are very low. For carcinogenic effects, extrapolation of dose-response data to predict risk levels smaller than 10^{-3} (1 in 1,000) or 10^{-4} (1 in 10,000) is scientifically tenuous. However, individual risk levels of 10^{-5} (1 in 100,000) or even 10^{-6} (1 in 1,000,000) are usually of concern to the public and to the policymaker, especially when substantial numbers of people are exposed. To overcome this policy dilemma, scientists usually extend their risk models to predict risk levels as low as 10^{-5} or 10^{-6}, incorporating "conservative" assumptions to avoid underestimating risks. However, the consequence of this conservatism is that standard procedures overestimate risk by an unknown (but potentially large) factor. The uncertainties in hazard assessment are usually compounded by uncertainties in exposure assessment, and by variability in exposure among different segments of the population.

B. Specific Problems Arising at Hazardous Waste Sites

Risk assessments for exposure to chemicals at hazardous waste sites are subject to a number of additional uncertainties, primarily arising from complexities in exposure assessment.

In most cases, a number of different chemicals are present at the site. Their quantities, distribution, and even their identities are usually unknown at the outset and have to be estimated from a limited number of sample measurements. It is now recognized that no hazardous waste site is totally secure, and a large number of environmental pathways by which chemicals may migrate from the site may have to be considered. However, since migration is often very slow, it is rarely possible to measure the exposure of off-site populations directly, and it is necessary to rely on models of chemical migration. Constructing such models requires knowledge of the characteristics of the chemicals and of the local environment (soils, groundwater, surface water, etc.) that is rarely available in sufficient detail for precise modelling. Although models sometimes can pinpoint the need for additional monitoring data (e.g., when calculations of volatilization rates suggest that concentrations of chemicals in air may be high enough to be of concern), the long time-scale of many hydrogeological processes means that models of transport in groundwater are often practically unverifiable. For these reasons, <u>assessments of exposure to chemicals migrating from hazardous waste sites are rarely accurate to better than order of magnitude</u>. Unless precise monitoring data are available in a specific case, it is usually a waste of resources to develop elaborate models of exposure and risk, and it is scientifically misleading to present the

results as though they were precise and accurate predictions of exposure and risk.

C. Procedures Used in the Report

The general procedures for risk assessment used in this report have been developed by scientists and engineers at Clement from experience at a number of hazardous waste sites. First, we review the information available on the chemicals present at the site and develop a short list of those giving rise to the greatest concern, on the basis of quantities present, toxicity, and propensity for migration leading to significant exposure. (In the case of Vickery, PCBs were prespecified for primary attention because of their special regulatory status under the Toxic Substances Control Act, but we identified 14 other chemicals for attention based on their presence at the site in substantial quantities and their toxicity (Table 2).) Second, we review information on the disposition of the chemicals at the site, and on engineering, hydrogeological, and other factors that determine the potential for these chemicals to migrate through or away from the site. Instead of attempting to construct detailed models of the transport of all chemicals by all possible pathways, we identify a limited number of pathways for specific attention. (In the case of Vickery, we identified volatilization and groundwater infiltration as the pathways of primary concern, but also recognized that the potential for surface water runoff had not been fully investigated and recommended additional sampling to verify its lack of importance.)

Third, for each transport pathway so identified, we develop scenarios under which the greatest exposure to human populations might result, and construct simplified models of transport and exposure for each. (In the case of Vickery, we constructed four scenarios for exposure, excluded two of them as unimportant on the basis of monitoring data, and developed simplified models for the other two.) Fourth, we combine exposure predictions from the simplified models with information on the size and distribution of populations at risk. (In the case of Vickery, we consider the workers on-site and the scattered population residing within 5 km of the site to be the primary groups at risk from exposure to airborne emissions.) Fifth, we review the remedial options under consideration for the site and evaluate the degree to which risks calculated for each scenario would be reduced (or increased) under each remedial option. Finally, we consider short-term risks that arise during, and as a result of, the remedial work.

Each of the steps listed above is highly site-specific and is usually limited more or less severely by the nature of the data available. The risks identified and estimated in each section of the assessment are not only subject to substantial scientific uncertainty, but also vary widely in nature and extent. For example, some risks may be of acute chemical exposure or mechanical accident to remedial workers; others may be expressed as a less-than-adequate margin of safety for a limited group of on-site workers or off-site residents; and

others may be expressed as a possible cancer risk of 10^{-6} or 10^{-5} to another group of persons. Overall characterization of the risks posed by the site under various remedial options involves weighing short-term risks against long-term risks, very uncertain risks against less uncertain risks, and risks to occupationally exposed workers against risks to third parties. We regard it as impossible to derive a single numerical characterization of the nature and magnitude of the risks posed by a site. Instead, we give a verbal characterization of the risks arising under each exposure scenario ("negligible," "low," "moderate-to-high," etc.) and an overall characterization of the risks posed by the site in the same terms. Since these characterizations involve some subjective judgment, we present them separately for each type of exposure, so that policymakers who are inclined to assign different relative weights to them can do so. For example, at the Vickery site, it is our judgment that long-term risks resulting from chemical exposures are small and uncertain, whereas short-term risks arising from remedial work under some remedial options are both larger and more certain to occur. However, for those who may disagree with either of these judgments, our assessment of risks of different types is disaggregated in Table 5, and its basis is explained in the text of Sections III and IV of this report.

III. TOXICITY OF PCBs AND OTHER CHEMICALS IDENTIFIED AT THE OLD SITE

A. Toxicity Assessment for PCBs

The toxicity of PCBs is evaluated in this section, which includes a survey of the literature, consideration of the difficulties of such evaluations, a review of key studies, and a discussion of limitations to such studies. Margins of safety are considered in the final segment of this section.

1. Literature Survey

Scientific data on the toxicity of PCBs and of their contaminants and related compounds have been reviewed in many papers, books, and scientific reports. The reference list includes 25 of these scientific reviews published between 1972 and 1983. We have reviewed most of the available scientific information summarized in these reviews and published in recent scientific journals. The scientific literature on the toxicity of PCBs is voluminous and complex, and a wide variety of toxic effects has been documented in studies of varying quality and conclusiveness. Appendix B to this report summarizes the toxicity studies which we judge to be the most relevant to assessing the possible hazards posed by the current contamination of the OLD site with PCBS.

Data on effects of PCBs in humans are derived from three main sources.

- In studies of workers exposed to relatively high levels[1] of PCBs in the workplace, the most consistent effect reported is a skin disease known as chloracne. Other effects reported less consistently or less frequently include induction of hepatic microsomal enzymes, other changes in liver function, neurological and behavioral symptoms, digestive disturbances, eye irritation, and respiratory impairment (NIOSH 1977, Warshaw et al. 1979). Three studies have suggested possible excess frequencies of cancer in exposed workers (Bahn et al. 1976, Brown and Jones 1981, Bertazzi et al. 1981), but these studies were limited by small sample sizes, were mutually inconsistent, and are not generally regarded as conclusive.

- Two incidents have been reported in which large numbers of people were poisoned by consumption of cooking oil contaminated with PCBs and with their thermal decomposition products. These incidents took place in Japan, where the disease is known as "Yusho" (Kuratsune et al. 1972, 1976), and in Taiwan (Chang et al. 1980). The symptoms included long-lasting skin lesions similar to those observed in chloracne, but also included abnormal pigmentation of the skin, menstrual disorders, liver damage, neurobehavioral impairment, minor birth abnormalities, respiratory symptoms, and immunological impairment.

- In the general population, exposure to PCBs (as measured indirectly by residues of PCBs in the blood or body fat) has been statistically correlated with elevated blood pressure (Kreiss et al. 1981), elevated levels of liver enzymes and serum triglycerides (Baker et al. 1980), premature births (Wassermann et al. 1982), missed abortions (Bercovici et al. 1980), and various types of cancer (Unger and Olsen 1980). However, these effects were also associated with exposure to DDE and other chemicals, and the results are ambiguous.

Experimental studies in animals have revealed a wide spectrum of adverse effects of PCBs when administered under appropriate conditions. Some of these effects are closely parallel to effects observed in humans. For example, PCBs induce hepatic microsomal enzymes in a number of animal species, and cause

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[1] "High" occupational levels are 200-2000 $\mu g/m^3$ plus skin contact.

skin lesions in rhesus monkeys that closely resemble those observed in human chloracne. Some effects observed in animals, such as immunosuppression and gastric lesions, have been reported only infrequently or equivocally in humans. Other effects have not been observed in humans, such as the induction of birth defects in dogs and swine. Several studies have indicated that PCBs can cause or enhance the induction of liver cancer in rats and mice.

2. Problems in Hazard Assessment

Assessment of the hazards posed by PCBs is made very complex by a number of factors.

- PCBs are complex mixtures and a number of commercial products have been sold and used which varied substantially in composition. It has long been known that these products differ in toxicity to at least a moderate degree. Some of the most serious toxic effects in animals (e.g., reproductive impairment in rhesus monkeys and liver cancer in rats) have been demonstrated only in experiments with mixtures (Aroclors 1248 and 1260) that were never used commercially in large quantities.

- PCBs are subject to contamination by highly toxic impurities, of which the most important are polychlorinated dibenzofurans (CDFs). These impurities were present at only trace quantities (1-5 ppm) in the commercially pure PCB mixtures, but can be formed in service under certain conditions involving high temperature and the presence of oxygen. There is little doubt that the high toxicity of the PCB mixtures in the "Yusho" incidents in Japan and Taiwan was due primarily to CDFs formed in service in this way.

- The results of studies with PCBs, especially in exposed human populations, have often been inconsistent, and some positive results have not been confirmed when the studies were repeated under similar conditions.

- Some studies have been of poor quality and can be accorded little weight in hazard assessment.

- The effects of PCBs often appear widely different in different species of animals. For example, species such as the mink and the rhesus monkey appear to be extremely sensitive to certain PCB mixtures, whereas the more conventional test species such as rats, mice, and dogs appear much less sensitive.

- The human studies have been limited in scope and duration, and they have not yielded conclusive evidence about the potential of PCBs to cause reproductive impairments or cancer in exposed populations.

For these reasons, selection of scientific studies to serve as the basis for risk assessment for PCBs and CDFs is unusually difficult. On the one hand, it can be argued that studies in animals provide evidence for serious toxic effects at very low exposure levels. In particular, the toxic effects observed in rhesus monkeys are parallel to those in humans exposed to PCBs either in the workplace (chloracne) or via contaminated food (Yusho). Studies in rhesus monkeys have revealed serious effects on reproduction and neurobehavioral development at low exposure levels. On the other hand, it can be argued that chloracne and Yusho are probably caused primarily by CDFs, that the monkeys were exposed to a mixture (Aroclor 1248) to which there has been very little human exposure, that reproductive and neurobehavioral effects were not reported in Yusho victims, and that more conventional test animal species have not shown significant effects at low exposure levels. With reference to human studies, it can be argued that studies of workers exposed to PCB mixtures made in the United States have not unequivocally shown significant effects,

except for chloracne at high exposure levels and inconsistent effects on liver function.

These arguments have been used by Ecology and Environment (1981) and Drill et al. (1982) to support the conclusion that risks posed by PCBs at low levels in the environment are insignificant. However, this conclusion has not yet been adopted as a consensus view by other scientific reviewers or by regulatory agencies such as the U.S. Environmental Protection Agency (USEPA 1980). Pending resolution of this scientific debate, for the purposes of this risk assessment we place weight on the scientific studies that suggest that PCBs may be toxic at low exposure levels. However, we recognize that the relevance of these studies has been questioned and that our assessment may be unduly conservative.

To serve as the basis of our risk assessment, we have selected three types of toxic effect of PCBs that are judged most likely to be relevant at low exposure levels. (Appendix C explains why other types of toxic effect are considered less relevant). The following section summarizes data on these toxic effects, and assesses their importance at low dose levels.

3. Assessment of Key Studies

a. Chloracne

Chloracne is a disfiguring skin disease that has been associated with exposure to PCBs, both in the workplace (NIOSH 1977) and through accidental contamination of food (Kuratsune 1972, 1975; Chung et al. 1980). The incidents of food contamina-

tion are complicated by the presence of high levels of CDFs and other contaminants, so the following assessment is based on dose-response data obtained through studies of workplace exposure to relatively uncontaminated PCBs.

NIOSH (1977) reviewed the data on the occurrence of chloracne in workers exposed to PCBs, and concluded that chloracne was generally associated with PCB concentrations in the blood of 200 ppb or greater. Fischbein et al. (1979) reported a high incidence (45-55%) of skin lesions (not limited to chloracne) in workers in a capacitor manufacturing plant whose average plasma concentration of PCBs was 172 ppb. Because of wide variability in blood concentrations and uncertainty in diagnosis, these and other reports on occupational exposure do not clearly specify a no-observed-adverse-effect-level (NOAEL) for chloracne. However, Humphrey (1977) reported no significant increase in skin disorders in groups of people exposed to PCBs via fish, with mean blood levels of PCBs ranging from 46 to 82 ppb, and individual levels ranging up to 366 ppb. Recognizing the possibility that chloracne may be caused primarily by CDFs, we will use these data as the basis for the assumption that 80 ppb in the blood is a NOAEL for skin effects of PCBs in healthy adults exposed to environmental residues of PCBs. This figure is selected as the highest average level for a group of people with no overtly detectable effects.

This blood concentration can be converted to equivalent PCB intake, using the results of the study by Humphrey (1977),

which indicated that a blood concentration of 58 µg/liter corresponded to an average daily intake of about 130 µg/day. Assuming that the relationship between intake and blood PCB level is linear, an average daily intake of PCBs of about 180 µg (80 x 130/58) would be a NOAEL.

b. Reproductive and Neurobehavioral Effects

Allen and his coworkers have reported a series of studies of the effects of PCBs (Aroclor 1248) on the health and reproductive performance of rhesus monkeys (Allen et al. 1975, 1979; Bowman et al. 1978, 1981). At relatively high concentrations in the diet (10 and 5 ppm), PCBs severely impaired reproduction, and the few infants who were born alive showed signs of poisoning, some of which closely resembled the signs of chloracne in man. Similar (but less severe) effects were observed in monkeys exposed to PCBs at 1 and 0.5 ppm on the diet. In the most recent study (Bowman et al. 1981), monkeys exposed to PCBs at 0.5 ppm in the diet for 3 days per week throughout pregnancy and nursing bred more or less normally, but the infants were underweight at birth, developed minor signs of poisoning (hyperpigmentation of skin) during the nursing period, were hyperactive and showed impaired learning ability when tested at 6 months of age. This average dietary concentration of 0.21 ppm is the lowest dose level at which significant adverse effects of PCBs have been reported in experimental animals. This dietary concentration is equivalent to a daily dose of

0.011 mg/kg body weight/day in the monkeys, which would correspond to a daily intake of about 700 µg/day in adult humans.

c. Carcinogenic Effects in Animals

Several studies have shown that PCBs are carcinogenic in rats and mice, causing increased incidence of liver tumors when administered to the animals at high levels in the diet for most or all of their lifetimes. However, only two of the studies were conducted systematically enough to serve as the basis for risk assessment. In a study by Kimbrough et al. (1975), lifetime exposure of rats to a diet containing 100 ppm Aroclor 1260 led to a high incidence (26/184 or 14.3%) of carcinomas of the liver, and a very high incidence (144/184 or 78.3%) of neoplastic nodules. In a smaller study by the National Cancer Institute (1978), lifetime exposure of rats to a dietary concentration of 100 ppm Aroclor 1254 led to a much smaller incidence of liver tumors (2/48 carcinomas and 3/48 neoplastic nodules or adenomas, pooling data for males and females). Although the differences in tumor frequency between the two experiments are large, the biological difference is less important, because the rats in the NCI study also had a high incidence of "hyperplastic nodules," a condition regarded by many pathologists as an earlier stage in the development of liver tumors. Thus, the main difference appears to have been that tumors developed more rapidly in the Kimbrough experiment than in the NCI experiment.

Several writers have used the data of Kimbrough et al. (1975) as the basis for low dose risk assessment. The most comprehensive assessment was that of Crump and Masterman (1979), who calculated that a dietary concentration of 6.9 ppb would correspond to a lifetime excess risk of 10^{-5} (1 in 100,000) for hepatocellular carcinomas, and that a dietary concentration of 0.63 ppb would correspond to a lifetime excess risk of 10^{-5} for neoplastic nodules. If it is assumed that humans would respond similarly at the same dietary concentrations, then a lifetime excess risk of 10^{-5} would correspond to daily intakes of about 10 ug and 1 ug, respectively.[1]

It should be emphasized that these estimates of possible carcinogenic risk are tenuous. They involve extrapolation from an effect observed in rats at a very high dose level to predict effects in humans at doses 100,000 times smaller. Although this extrapolation follows EPA's current procedures for carcinogenic risk assessment, these procedures are intended to be conservative, and are currently under review. PCBs are known to act by "promoting" the effects of other carcinogens (Nishizumi 1976, 1979, Preston et al. 1981). However, these effects have been studied only at high doses, and there are no accepted procedures for risk assessment in these circumstances. Some recent writers have proposed that thresholds

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[1]Similar results were presented by NAS (1977) and EPA (1980). The results are sensitive to the assumption that is made about the way in which doses should be scaled between species (Nisbet 1981), but the assumption made in the text conforms to EPA's current practice.

exist for the action of cancer promoters and that safe levels of exposure can be established by applying a margin of safety to a no-observed-effect-level. However, this approach cannot yet be applied to PCBs because no experiment has been conducted to establish a NOAEL for cancer promotion.

4. Limitations of the Studies

Table 1 summarizes the three types of toxic effects that are considered in our risk assessment. The column headed "Comments" indicates that each of the three sets of data has substantial limitations and that there are several reasons to question their direct applicability to prediction of human risks. Nevertheless, they include the best-documented subchronic effect in humans and the two effects of greatest concern that have been reported in experimental animals. For these reasons we use them as the basis for risk assessment, while emphasizing that the limitations pointed out in Table 1 may lead to overestimation of risks.

5. Margins of Safety

For each of the three types of toxic effect considered in this risk assessment, some information is available on dose-response relationships. That is, for at least one level of dosage, effects have been measured in animals or humans under conditions of prolonged exposure. Accordingly, for any hypothetical situation in which humans would be exposed for long periods to lower levels of PCBs, "margins of safety" can be calculated.

TABLE 1

TOXIC EFFECTS CONSIDERED IN RISK ASSESSMENT

Effect	Dose-Response Data	Comments
Choracne		
Occupational skin disease, liver and neurobehavioral symptoms in some cases	Associated with blood levels over 200 ppb; no effects in groups with 46-82 ppb	Reported effects variable; may reflect misdiagnosis and effects of CDFs; not necessarily the most sensitive indicator of long-term exposure to PCBs
Reproductive Impairment		
Failure to conceive, skin lesions, retarded development, learning deficits	Minor effects at lowest dose tested, about 11 µg/kg/day to mothers	Demonstrated only in rhesus monkeys; other species less sensitive Demonstrated only with Aroclor 1248; possible role of CDFs; small sample
Cancer Induction		
Liver cancer in rats in one good and several unsatisfactory experiments; negative or marginal effects in others	Tested only at very high dose, 100 ppm in diet or about 8 mg/kg/day	Demonstrated only with Aroclor 1260; weak or equivocal effects with Aroclor 1254; primarily cancer promoter

Figure 1 presents a graphic description of the relationship between levels of intake and margins of safety. The left-hand column indicates (on a logarithmic scale) daily intakes of PCBs in μg/day and indicates the equivalent long-term average intake levels at which toxic effects have been observed. The middle column characterizes the margin of safety at various lower dose levels. For example, an average daily intake of about 100 μg/day would be slightly greater than half the no-observed-effect level (NOAEL) for chloracne, and about one-seventh that caused adverse effects in monkeys. These margins of safety would normally be considered inadequate, because they would not allow sufficiently for variations in sensitivity within the human population, and for differences in sensitivity between human and animal species. On the other hand, an average daily intake of about 1 μg/day would be about 1/180 times the NOAEL for chloracne, and about 1/700 times the lowest observed effect level in monkeys. These margins of safety would ordinarily be considered adequate to ensure safety to exposed populations, even under conditions of long-term exposure. If average daily intakes were kept below 1 μg/day, the margins of safety for these effects would ordinarily be considered at least ample.

The right hand column in Figure 1 shows conservative estimates of the possible cancer risks that might result from long-term (lifetime) intake of PCBs at the indicated rate. For example, continuous ingestion of 1 μg/day of PCBs throughout

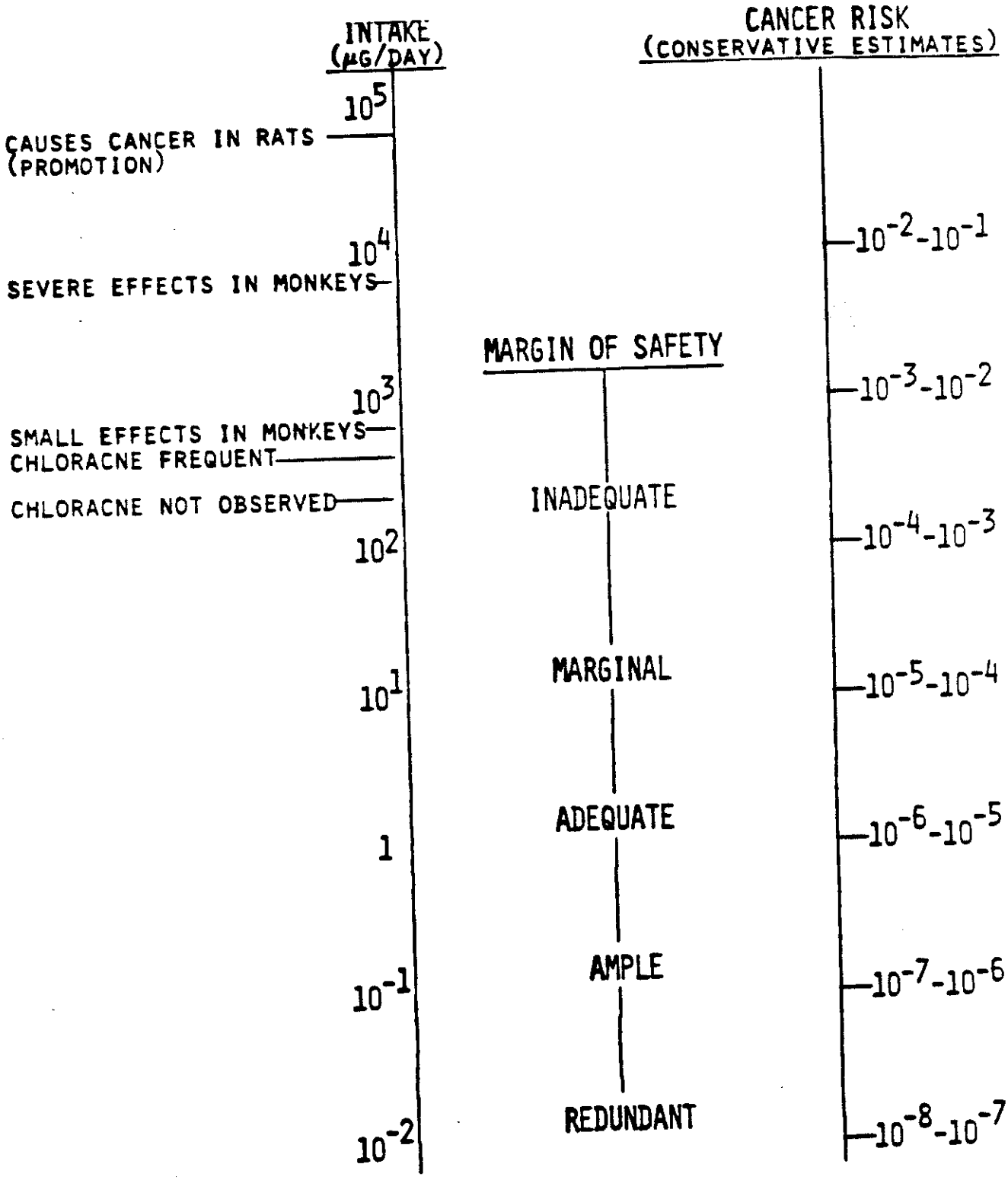


FIGURE 1

MARGINS OF SAFETY FOR LONG-TERM INTAKE
OF PCBs AT VARIOUS DAILY DOSE LEVELS

life might, under the assumptions of the calculations, lead to cancer risks in the range of 10^{-6} and 10^{-5} (1-10 per million). For the reasons explained above, these estimates are tenuous and are designed to be conservative.

The risk assessments illustrated in Figure 1 are made specifically for continuous intake of PCBs for long periods (lifetime intake for cancer risks, exposure for months or years for other types of effect). If exposure is for short periods or is intermittent, margins of safety would be correspondingly higher.

B. Toxicity of Other Contaminants

In addition to PCBs, several other priority pollutants have been detected in the sludges and aqueous layers of the ponds at the OLD site. Since these chemicals are found in association with PCBs and will be subjected to the same remedial actions, we have considered briefly the potential for human exposure and health risks that may arise from their presence (see Sections IV D and IV F below). However, since these chemicals are not the primary subject of this risk assessment, we have relied on secondary sources for toxicity assessment. Specifically, we have utilized the Water Quality Criteria Documents for these pollutants as issued by EPA in 1980. These documents included detailed reviews of toxicity information available at that time and risk assessments for both carcinogenic and noncarcinogenic effects.

The pollutants identified in substantial quantities at the site are the following:

Toluene

Ethylbenzene

Chloromethane

Dichloromethane

Chloroform

Carbon tetrachloride

1,2-Dichloroethane

1,1,1-Trichloroethane

Trichloroethylene

Tetrachloroethylene

Chlorobenzene

o-Dichlorobenzene

m-Dichlorobenzene

p-Dichlorobenzene

Table 2 lists for each of these chemicals the water quality criteria numbers calculated by EPA, and the corresponding daily intakes by an average person. For carcinogenic chemicals, these intakes are intended to represent an exposure level that, under conservative assumptions, would not lead to a lifetime risk of cancer greater than 10^{-5} (1 in 100,000). To place this risk level in perspective, the workforce at the OLD site incudes 55 workers, and about 850 other persons who live within about 5 km of the site. Assuming long-term exposure for all these persons, a life-time risk of 10^{-5} for each person would

TABLE 2

CRITERIA NUMBERS AND DAILY INTAKES FOR 13 CHEMICALS FOUND AT THE OLD SITE[a]

Chemical	Carcinogenic Effects		Noncarcinogenic Effects	
	Water Quality Criterion (10^{-5} Risk) (µg/liter)	Corresponding Daily Intake (µg/day)	Water Quality Criterion (µg/liter)	Corresponding Daily Intake (µg/day)
Ethylbenzene			1,400-8,500	1,600-9,500
Chloromethane			7,500	7,500
Dichloromethane			12,400	24,800
Chloroform	1.90	3.85		
Carbon tetrachloride	4.0	8.5		
1,2-Dichloroethane	9.4	18.9		
1,1,1-Trichloroethane			18,400	37,500
Trichloroethylene[b]	27	55.9	18,300	38,000
Tetrachloroethylene	8.0	17.6		
Chlorobenzene			20	41.3
o-Dichlorobenzene			400	945
m-Dichlorobenzene			400	945
p-Dichlorobenzene			400	945

[a]An Ambient Water Criterion was not formulated by EPA for toluene, but the toxicity of this chemical is low and comparable to that of ethylbenzene (NAS 1980).

[b]A criterion for noncarcinogenic effects is also given because the study used as a basis for the criterion for carcinogenic effects had several major technical problems which make the results subject to question. This latter criterion, however, was the one recommended by EPA in 1980.

correspond to a probability of about 9×10^{-3} (less than 1 in 100) that one excess cancer would occur in this population over a 70-year period. For the average person in the U.S. population, the probability of dying of cancer is about 0.24 (1 in 4); an excess lifetime risk of 10^{-5} would increase this by a factor of 1/24,000 (0.004%). Table 3 lists the lifetime risk of death from a number of causes. The lifetime risk of 10^{-5} is about one-third of that of being killed by lightning, and is similar to the excess risk imposed by driving a car for about 20 minutes, or by smoking one cigarette every 3 years. For noncarcinogenic chemicals, the intakes listed in Table 2 are intended to provide an adequate margin of safety below the lowest levels reported to cause toxic effects in humans or animals. For the purposes of this report, we assume that the rates of intake tabulated in Table 2 would correspond to an adequate margin of safety for long-term exposure to these chemicals. For PCBs, an adequate margin of safety for noncarcinogenic effects corresponds to a lifetime risk of 10^{-5} or less for carcinogenic effects (Figure 1). For other chemicals, Gaylor (1983) has shown that the procedures used to establish an adequate margin of safety would generally ensure that cancer risks would be less than 10^{-4}. Thus, although there is no exact correspondence between the numbers obtained by the two procedures, the intakes listed in Table 2 may be somewhat more protective for carcinogenic effects than for noncarcinogenic effects.

TABLE 3

LIFETIME RISKS OF DEATH FROM VARIOUS CAUSES
IN THE GENERAL U.S. POPULATION

Cause of Death	Risk Over Lifetime of 70 Years
Vaccination for small pox (once in a lifetime)	3.0×10^{-6}
Lightning	2.8×10^{-5}
Nuclear power	3.2×10^{-5}
Home appliance	3.5×10^{-5}
Natural disaster	7.0×10^{-5}
Airplane accident (1 transcontinental flight/year)	2.1×10^{-4}
Electrocution	3.5×10^{-4}
Medical X-rays	7.0×10^{-4}[a]
Accidental poisoning	9.1×10^{-4}

[a]Risk of cancer, not necessarily of death

SOURCES: Fischhoff et al. 1982 and Wilson 1978

IV. EXPOSURE SCENARIOS AND RISK ASSESSMENT

A. Transport of PCBs in Air

The first exposure scenario to be explored is the volatilization of PCBs from the ponds or other contaminated areas at the site, leading to exposure via inhalation of workers on-site or of persons in the general population resident downwind. The potential for such exposure has been investigated directly by ERT, who collected three series of ambient air samples for analysis for PCBs. The first series of samples was collected at five sites around the perimeter of the OLD facility and utilized polyurethane foam adsorbents (USEPA's recommended method). Five or six samples were collected at each site. PCBs were not detected in any sample at detection limits between 0.05 and 0.1 $\mu g/m^3$ (ERT draft report of April 22, 1983). The second series of samples utilized personal monitors carried by workers at the facility with Florisil and polyurethane foam adsorbents (NIOSH method P&CAM 253). Prior to the initiation of remedial work, two personal monitor samples and three area samples were collected from the skimming and oil recovery areas. PCBs were not detected in any sample at detection limits of about 0.15 $\mu g/m^3$ (ERT letter of May 26, 1983). The third series of area samples was collected at six sites around and downwind of the facility and also utilized NIOSH method P&CAM 253. PCBs were not detected in any sample at detection limits of about 0.5 $\mu g/m^3$ (ERT report of July 1983).

Assuming typical breathing rates (not exceeding 10 m^3/person during an 8-hour workday, or 20 m^3/person during a 24-hour period), these data suggest upper limits of 1.5-5 µg/day for intake of PCBs by inhalation for workers on site, 1-2 µg/day for persons at the fenceline, and progressively less for persons at increasing distances from the facility. Since PCBs were not detected in any sample, it is unlikely that actual intakes would approach these figures, which are based on the detection limits for the analytical methods. Accordingly, we conclude that risks resulting from exposure by inhalation under present conditions would be negligible. Under all remedial options, the potential for volatilization losses would be reduced, probably by several orders of magnitude, since PCBs would be removed from surface layers, fixed in a solid matrix, and/or removed from the site. (Transitory exposures by inhalation during remedial work will be considered in Scenario E).

B. Transport of PCBs in Surface Water

The second exposure scenario to be explored is the transport of PCBs off-site in surface water runoff, with subsequent exposure to consumers of fish downstream. PCBs were detected at trace levels (22 ppb) in one water sample from a stormwater retention ditch on the site in ETC's Phase III sampling. However, this ditch does not drain off-site, and all surface water runoff from potentially contaminated areas of the site either will be analyzed for PCBs prior to discharge or will be collected and pumped into the lagoons. To investigate the possibility

that PCBs from the site might nevertheless have migrated into off-site surface water, samples were collected from the the two principal surface water channels drying the vicinity of the site, Raccoon Creek and Little Raccoon Creek. PCBs were not detected (detection limit, 10 ppb) in water samples taken downstream from the site in either creek (ETC's Phase VI). To improve the sensitivity of this result, samples of water and sediment were taken in water and sediment in Raccoon Creek in July 1983; again PCBs were not detected in any sample at a detection limit of 10 ppb (ETC's Phase VIII). The most significant of these negative findings was in sediment immediately downstream from the site. Since PCBs are usually partitioned between sediments and water in a ratio of 10,000:1 or more, this indicates that PCB levels in the water of Raccoon Creek are unlikely to exceed about 1 ppt (parts per trillion). Even if present at this level, PCBs would be diluted downstream as the creek flowed into larger bodies of water and would pose a negligible risk to downstream consumers of fish.

C. Transport of PCBs in Groundwater

The third scenario for human exposure to PCBs migrating from the site is the possibility that PCBs might move through the soil in percolating groundwater, leading to human exposure either to water pumped from subsurface aquifers, or to surface waters downstream. Although the site is favorably situated on top of a 38-foot layer of relatively impervious clay, there is a theoretical possibility that PCBs will slowly migrate

downwards to the underlying aquifer, or horizontally though surficial soil layers to contaminate surface waters downgradient. These possibilities will be explored semiquantitatively in this section.

We note, first, that PCBs in the existing lagoons are found primarily in oily sludges. Although the partition coefficient for PCBs between these sludges and the aqueous phase in the lagoons has not been measured, it is likely to be at least 10^6 (reflecting the low solubility of PCBs in water, of the order of 50 ppb). Thus, with high concentrations of PCBs in the sludges of the order of 50-200 ppm, it is unlikely that the initial concentrations of PCBs in water percolating into the clay would exceed 0.1 ppb (100 parts per trillion). Even if undiluted, such a concentration would pose negligible risk to human health, since a person drinking 2 liters of water containing 0.1 ppb of PCBs per day would ingest only 0.2 μg/day. PCBs at such a concentration would only be of concern if they migrated into surface water and were reconcentrated in fish.

We note, second, that there is evidence that sulfuric acid in the lagoons has reacted with materials in the clay lining to form a highly impervious layer of calcium sulfate (gypsum). Although this layer is expected to retard percolation from the lagoons into the unaltered clays, we have no information on its characteristics and therefore have not considered it in our models. This is appropriate for the situation arising under some of the remedial options, in which the gypsum layer

would be at least partly broken up. However, for the unremedied situation, the omission of the impervious layer from our models will lead to substantial overestimation of the potential for migration.

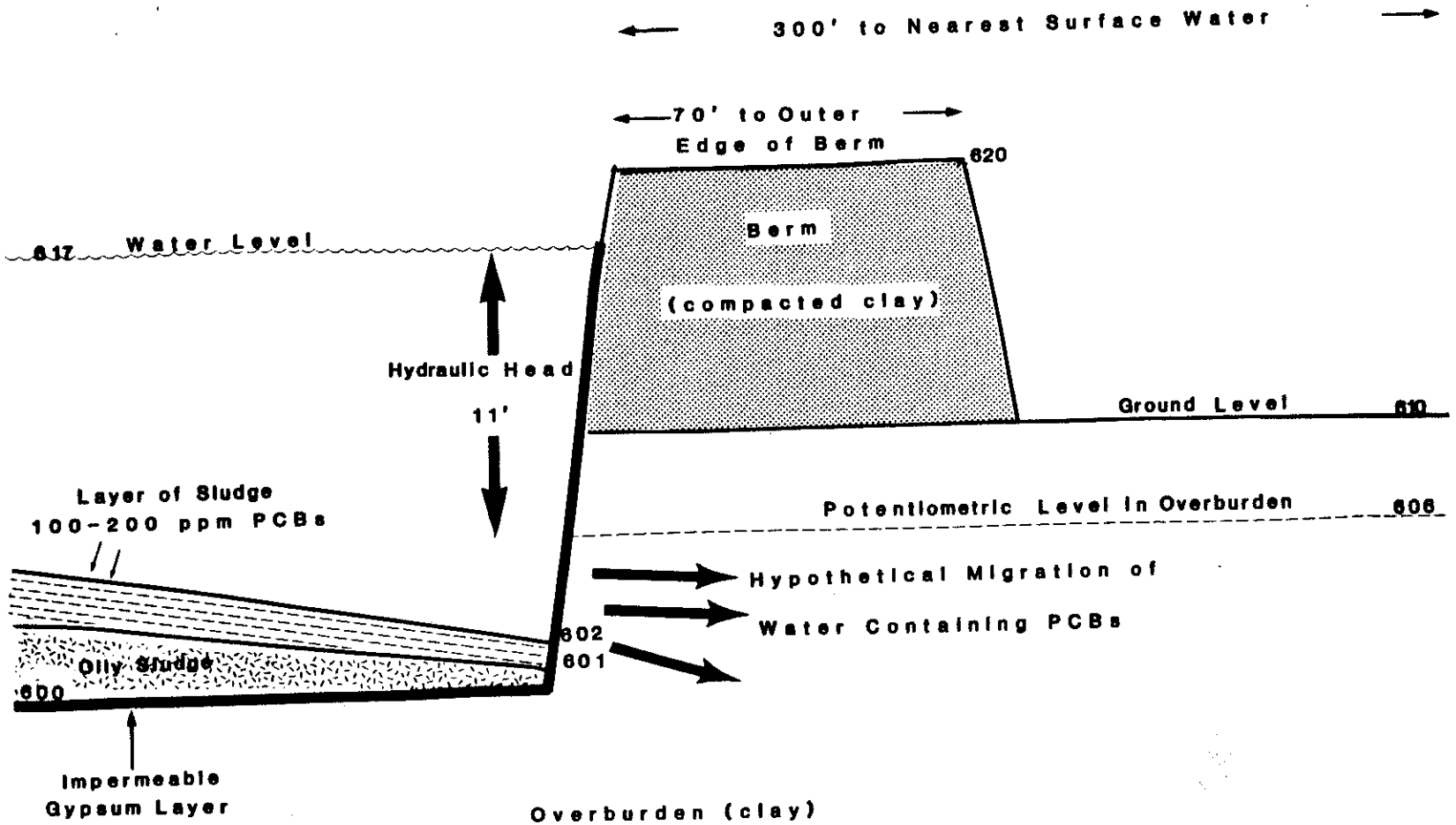
The potential for downward migration of aqueous-phase materials from the site has already been calculated by Golder Associates (Exhibit III to CWM's presentation of July 28, 1983). Using the measured vertical hydraulic gradient of 0.33 (12'/36'), the vertical permeability of the clay of 2×10^{-8} cm/s (average of laboratory measurements), and an assumed porosity of 0.1, they estimated the average rate of downward percolation though the overburden as about 0.02 m/yr. (0.07 ft/yr). Neglecting retardation in the gypsum layer, it would than take about 400 years for aqueous phase materials from the lagoons to reach the underlying aquifer. However, PCBs are strongly adsorbed to the surfaces of clay particles, and PCBs would not reach the underlying aquifer until the overburden were saturated with them. Under the conditions assumed in Golder's model, the volume flow of water through a 1 m^2 horizontal cross-section would be about 0.002 m^3/year, and the mass flux of PCBs (at an initial concentration of 0.1 ppb) would be about 0.2 µg/year. Assuming a typical partition coefficient of 10^4 between clay and aqueous phase, the equilibrium concentration in the clay would be of the order of 1 ppm. The time required for PCBs to saturate the overburden to this level and to reach the aquifer would then be of the order of 100 million years. Even after this

time, the aqueous concentration of PCBs reaching the aquifer would still be of the order of 0.1 ppb and would be further diluted by the water flowing through the aquifer.

A somewhat higher potential for exposure may arise from horizontal percolation though the surficial layers, since the horizontal permeability of the clay is much higher than its vertical permeability--of the order of 10^{-7} cm/s and perhaps locally as high as 10^{-6} cm/s (Golder Associates, report of June 1983). In our judgment, the greatest potential for such movement is at the north end of lagoons 4 and 5, where a layer of sludge containing PCBs is present in the lagoons between 0 and 8 feet below the level of the surrounding terrain, and is under a hydraulic head about 11' greater than that in the surrounding overburden. Accordingly, we have constructed a simple model of a scenario in which aqueous-phase materials containing PCBs would migrate horizontally though the soil under the berm at the north end of the lagoons, eventually emerging near the surface of the soil 20 m to the north (on-site) or into surface waters 100 m to the north (off-site). This scenario is illustrated diagramatically in Figure 2. Using a simplified one-dimensional model and assuming a linear hydraulic gradient, the calculation presented above for vertical migration can be adapted simply to cover horizontal migration. For transport 20 m though the berm, the hydraulic gradient would be 0.16, so that the average velocity of flow would be 0.05 m/yr for a permeability of 10^{-7}cm/s and 0.5 m/yr for local

FIGURE 2

Schematic Model of North End of Lagoon 5 to Illustrate Hypothetical Horizontal Migration of Water Containing PCBs



zones with permeability 10^{-6} cm/s. For transport 100 m to off-site drainage channels, the average velocity of flow would be 0.01 m/yr for a permeability of 10^{-7} cm/s and 0.1 m/yr for local zones with permeability 10^{-6} cm/s. Even under the last scenario, the worst case for human exposure off-site, it would take a period of the order of 100 million years for the PCBs to saturate the subsurface clays and to reach the off-site surface water channel. Even after this period, the aqueous concentration of PCBs reaching the surface channel would still be of the order of 0.1 ppb and would be further diluted as they mixed with the water in the channel and moved downstream.

Based on the above simple calculations, we judge the risks posed by migration of PCBs in groundwater from the lagoons as they exist at present to be negligible. Hypothetically, PCBs might also migrate through the overburden in an organic phase separate from the aqueous phase. If so, the initial concentration of PCBs would be higher and the time required to saturate the overburden would be lower. In our judgment, the nature of the sludge is such as to make the likelihood of non-aqueous phase migration unlikely, and this is supported by observational evidence, which revealed very slow percolation of aqueous-phase materials and no oily materials in a boring into a berm between the lagoons (Golder Associates, personal communication from G. Collison, July 1983). However, on the information available, the possibility of non-aqueous phase migration cannot be altogether excluded. For this reason,

we classify the risk of exposure resulting from groundwater contamination under present (unremedied) conditions as very low.

Under remedial option 1, sludges would be fixed in a solid matrix and reinterred in situ in lagoons 4 and 5. Although this may reduce the potential for leaching into aqueous-phase liquids, it is not certain that it would do so, since the partition coefficient of PCBs between oily sludges and water is already extremely high and that between the fixed sludges and water may not be higher. Also, option 1 will at least partly remove the protective gypsum lining of the lagoons. In the absence of a leachate collection system, the filled pond will eventually fill with water percolating through the clay cap (the "bathtub effect") and an outward hydraulic gradient will be re-established. Thus, in our judgment, remedial option 1 will probably not yield a net reduction in the potential for migration into groundwater.

Remedial options 2, 3, and 4 are intended to remove a large fraction of the PCBs off-site. However, they would also remove most of the other organic materials that are responsible for the partitioning of PCBs out of the aqueous phase. Hence it is not clear that the possibility for migration of PCBs into groundwater would be reduced.

Remedial options 1A, 1B, and 5 all involve fixing of the PCBs in a solid matrix and reinterring them in a secure cell with a leachate collection system. Under these options, the operation of the leachate collection system will suffice to

reverse the hydraulic gradient. Hence, any movement of PCBs in groundwater would be inwards rather than outwards, and the possibility for human exposure via groundwater movement will be negligible or zero.

Remedial option 6 would involve the transport of all contaminated sludges offsite, so the possibility for significant migration would be negligible or zero. (We assume that they would be disposed of at another site where environmental conditions are equally favorable).

D. Transport of Other Volatile Organics in Air

In addition to PCBs, a number of other volatile organic compounds (VOCs) have been detected in the sludges and aqueous layers of the lagoons at the OLD site. Some of these chemicals are present in larger concentrations then PCBs, are more volatile, and are less prone to be trapped in the oily surface layer of the lagoons. Accordingly, we have calculated the potential for exposure to these materials by inhalation, both on-site and off-site.

Preliminary engineering estimates of volatilization of VOCs from the lagoons at the OLD site were presented by ERT (Report of June, 1983), based on measurements of their concentrations in aqueous-phase samples collected and analyzed by Allied Analytical and Research Laboratories (Report of 28 June 1982). Subsequently, ETC performed analyses of samples collected from the lagoons in July 1983 (ETC, Phase VIII Report), and ERT revised its calculations of emission rates to reflect these

more current analyses (ERT, letter of July 12, 1983). Our calculations of potential exposure are based on these last estimates.

ERT's estimates of emission rates are dominated by those from lagoon 11, which accounted for about 80 percent of all the calculated emissions because of its large size and relatively high concentrations of certain VOCs. Table 4 shows the concentrations of VOCs in lagoon 11 in July 1983, and the corresponding estimates of emission rates under a typical set of summer weather conditions (wind speed 9.5 mph, the average speed for the year at Toledo, and temperature 22°C). Based on these calculations of emission rates, we have used a standard atmospheric diffusion model (neglecting degradation) to estimate the likely concentrations of these chemicals at various distances downwind under these weather conditions. For distances of 1, 2, and 5 km downstream, the model yields concentration estimates for total VOCs of 230, 190, and 69 $\mu g/m^3$, respectively. Of these concentrations, about 38% would consist of dichloromethane, 28% of chloromethane, 27% of chlorobenzene, and 4.4% of chloroform. Comparison with Table 2 indicates that these concentrations would be of concern primarily for chlorobenzene and chloroform. For both these chemicals, a person resident downwind and breathing 20 m^3/d of air might be subject to intakes in the range characterized as providing only a marginal to inadequate margin of safety (see Table 2 and Figure 1). Although these calculated intakes are not clearly hazardous (the calculated intake of

chloroform, for example, is similar to intakes by ingestion of drinking water from some chlorinated supplies), these estimates show at least the need for direct measurements of ambient concentrations, to verify or refute the predictions of the model.

We also considered exposure of on-site personnel to airborne emissions of VOCs from the lagoons. The worst case is probably for outdoor workers immediately downwind (east) of lagoon 11 under warm conditions (22°C) with a moderate wind (9.5 mph). Under these circumstances, we calculate that the effective mixing height for materials volatilizing from the lagoon is 9 m. Hence, if it is assumed that the materials are well-mixed, the average concentration would be about 520 $\mu g/m^3$. (This is only about twice the concentration calculated for 1 km downwind, because of the limited opportunity for hozizontal mixing close to an extended source.) For a worker working outdoors for an 8-hour workday, intakes would be similar to those calculated above for 24-hour residents farther downwind, and the same comments about margins of safety are applicable.

Under all remedial options, the potential for volatilization will be reduced, usually by several orders of magnitude. The options that provide for off-site removal of PCBs, or treatment of the contaminated sludges and off-site removal of the extracted materials, would also effect off-site removal of most of the other VOCs listed in Table 4. Other options that provide for

TABLE 4

CALCULATED EMISSION RATES OF VOLATILE

ORGANIC COMPOUNDS FROM LAGOON 11 AT VICKERY, OHIO

Chemical[a]	Concentration (µg/liter)	C/ MW[b]	Emission Rate[c] (g/s)
Chlorobenzene	14,400	1,352	1.43
Chloroform	2,620	243	0.26
Chloromethane	8,900	1,398	1.48
Dichloromethane	17,700	1,931	2.04
Toluene	1,210	126	0.13
1,1,1-Trichloroethane	1,050	102	0.11
Trichloroethylene	728	64	0.07

[a]Benzene, ethylbenzene, 1,2-dichloroethane and 1,1,2,2-tetrachloroethane were detected at small concentrations in the 1982 samples, but were below detection limits in the 1983 samples. Dichlorobenzenes (o-,m-, and p-) have been reported in the sludges, but not in aqueous samples.

[b]Concentration in µg/l divided by (molecular weight)$^{0.5}$. Volatilization rates of different compounds are expected to be in proportion to this quantity.

[c]Calculated using two-resistance theory for wind speed of 9.5 mph and air temperature of 22°C.

fixing of the sludges in a solid matrix and interring them in a secure cell will also greatly retard any losses of other VOCs, except perhaps option 1, in which water levels in the cells may be high and some VOCs may be able to migrate upwards to the surface. Thus, under all remedial options, we characterize the potential for volatilization of VOCs leading to significant exposure downwind as very low or negligible. However, it should be noted that most of the reductions in risk will not be effected until lagoon 11 is drained, unless emissions are reduced in the interim by reductions in inputs and hence in the pond inventory of these chemicals.

Conclusions on Long-Term Off-site Migration

Under all options for remedial action, the possibility for long-term movement of PCBs (and other volatile organic compounds) off-site and for significant long-term human or environmental exposure arising therefrom is very low or negligible. Under all options, therefore, potential risks are dominated by short-term exposures arising during the remedial work. These short-term risks are considered in the rest of Section IV.

E. Exposure of Remedial Workers to PCBs

During the remedial work, workers will be operating machinery and equipment that is handling substantial quantities of PCBs, and the workers may be exposed to PCBs both by inhalation and by dermal contact. This situation arises in all remedial work

at hazardous waste sites, and risks to workers are minimized as a matter of routine by the implementation of safety plans. A typical safety plan involves the specification and rigorous enforcement of safe operating procedures, use of protective clothing, monitoring of ambient concentrations and/or personal monitoring[1], medical surveillance of workers, and a contingency plan for accidents or emergencies. In our experience and in the experience of our consultants in occupational medicine, an adequate safety plan can reduce the possibility that workers will be exposed to hazardous levels of PCBs at a very low level. In any case, these risks are common to all remedial options and cannot be used to discriminate among them. The only exception to this generalization is that options 2 and 3 involve more handling of PCB-contaminated sludges and a solvent-treatment step, so that the possibilities for worker exposure are probably somewhat higher.

F. Exposure of Workers and Persons Off-Site to Volatile Organic Compounds Released During Remedial Work

Another possible route of exposure during remedial work is the release of other VOCs from sludges exposed to the air during excavation, transportation or other treatment. Some volatilization is to be expected, since the sludges contain

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[1]At the Vickery site, personal air monitors were used to collect 15 samples from workers sampling oil and skimming oil from the surface of the lagoons during remedial work in May-June, 1983. PCBs were not detected in any sample at a detection limit of 1 $\mu g/m^3$ (CWM data collected in May-June 1983). Dermal exposure is more difficult to measure, however.

substantial quantities of some of the materials listed in Tables 2 and 4, and the sludges are known to be highly odorous when first exposed to the air. The factors governing these transitory volatilization losses are so complex that it is impossible to calculate them a priori. Instead, we have recommended that airborne concentrations of VOCs should be measured in a pilot operation or in the first stages of a remedial program, and that these measurements should be used as the basis for designing a safety plan. At this stage in the risk assessment, it is our judgment that such exposures are of potential concern, because existing exposures to VOCs emitted from the surface of the lagoons may not provide an adequate margin of safety for long-term exposure (see above) and because such exposures are likely to increase, at least transitorily, during remedial work. Accordingly, we have recommended that design and execution of the remedial program and safety plan should take these exposures into account. This should include minimizing exposure of newly-excavated sludges to the air, minimizing worker exposure to vapors, avoiding work during hot periods of the year, monitoring ambient concentrations in the work area and at the fenceline, and operating a contingency plan to interrupt work if hazardous levels are approached. For the purposes of this risk assessment, we note that if these precautions are adopted, any risk arising from this source will be the same under any remedial option, and indeed would have arisen under any plan to close the lagoons at some time in the future.

G. On-Site Accidents

As in any operation involving the construction and use of heavy equipment, there is some risk of on-site accidents leading to injury or death of workers. We asume that these risks will be minimized by an effective safety plan, and will be the same under all remedial options (except options 2 and 3, which involve somewhat more on-site activity).

H. Transportation Accidents

The last category of risks to be considered is that arising from traffic accidents during the transportation of materials for off-site disposal. Some of the options would involve large-scale highway transportation in heavy vehicles. For example, option 6 would involve about 2,950 round trips to Emelle, Alabama, or about 4.8 million miles travel by heavy trucks. (This assumes transport of liquid sludges; if the materials were solidified before transport, total mileage would double to about 9.6 million miles). Option 4A (liquid sludges >50 ppm PCBs to Emelle) would involve about 2.3 million miles. Options 4B (semisolid sludges to Niagara Falls and Cincinnati) or 3 (solvent extracts to Chickasaw, Alabama) would each involve about 1.4 million miles. Option 2 (solvent extracts to Chickasaw, Alabama) wuld involve about 0.6 million miles. All options, including 1, 1A, and 1B, would involve some further transport of skimmed oils to Chickasaw, but this would involve less than 0.1 million miles in each case.

Large-scale highway transport on this scale involves substantial and predictable risks resulting from highway accidents. According to statistics compiled and published by the U.S. Department of Transportation,[1] large trucks are involved in about 451 accidents for every 100 million vehicle miles traveled, and these cause about 54 injuries and 5.3 deaths. In 1981, vehicles carrying hazardous materials were involved in 1,868 accidents in the United States, which caused 1,604 injuries, 202 deaths, and $31 million in property damage. Although specific data are not available on accidents involving hazardous materials per 100 million vehicle miles, it is noteworthy that the death and injury rates per accident are 9 and 7 times higher, respectively, than those for accidents involving all heavy trucks. Information on the frequency of chemical spills resulting from highway traffic accidents is collected by EPA's Office of Emergency Response, but even summary statistics are not released to the public.

Applying these statistics to the projected transportation needs for the proposed remedial options, option 6 would statistically be expected to result in about 22 accidents. Using statistics on all heavy truck accidents, these would be expected to result in 2 or 3 injuries and 0.25 deaths (i.e., 1 chance in 4 of a fatal accident). Using statistics on truck accidents involving hazardous materials, they would be expected to result

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[1] U.S. Department of Transportation, Bureau of Motor Carrier Safety, Accidents of Motor Carriers of Property, 1980-81 (August 27, 1982)), pp. 35-40, 51-52; National Highway Traffic Safety Administration, Large-Truck Accident Causation (July 1982), pp. III-4 and III-5

in about 18 injuries and 2 or 3 deaths. Although specific statistics are not available to serve as the basis for prediction, we assume that at least some of these accidents would lead to spills of cargo containing PCBs. In our judgment, these relatively tangible risks of death, injury, and chemical spills are much greater than the theoretical and conjectural risks of chemical exposure and health impairment discussed in previous scenarios. Accordingly, we classify option 6 as one which gives rise to moderate-to-high risks of injury and death. Since risks are more or less in proportion to vehicle miles traveled, we classify option 4A as of moderate risk, options 3 and 4B as of low-to-moderate risk and option 2 as of low risk associated with transportation.

V. CONCLUSIONS

Table 5 provides a summary characterization of the risks judged likely to arise under each of the remedial options, including the no-action option. In our judgment, the predominant risks are those due to transportation accidents, whose magnitude rises in proportion to the extent of off-site transportation. Risks due to exposure to volatile organic compounds other than PCBs (primarily chlorobenzene and chloroform) may be of some concern, especially during the remedial action, and it is recommended that these risks be carefully managed by means of a safety plan. Other risks, including all health risks due to exposure to PCBs, are judged to be either negligible or very low, provided that an adequate safety plan is implemented during remedial action.

TABLE 5

CHARACTERIZATION[1] OF RISKS ARISING UNDER VARIOUS REMEDIAL OPTIONS AT THE VICKERY, OHIO, SITE

Type of Risk	No Action	Remedial Option								
		1	1A	1B	2	3	4A	4B	5	6
Volatilization of PCBs from ponds	-	-	-	-	-	-	-	-	-	-
Surface water runoff of PCBs	-	-	-	-	-	-	-	-	-	-
Infiltration of PCBs into groundwater and transport off-site	0	0	-	-	-	-	-	-	-	-
Volatilization of other VOCs from ponds; on-site or off-site exposure	XX	0	-	-	-	-	-	-	-	-
Exposure of remedial workers to PCBs*	-	0	0	0	0	0	0	0	0	0
Release of other VOCs during remedial work; on-site or off-site exposure*	-	X	X	X	X	X	X	X	X	X
On-site accidents*	-	X	X	X	X	X	X	X	X	X
Transportation accidents	-	-	-	-	X	XX	XXX	XX	-	XXXX
Overall characterization	XX	0	-	-	X	XX	XXX	XX	-	XXXX

-, negligible (at least ample margin of safety);

0, very low (adequate margin of safety);

X, low (marginal margin of safety; 2-3 accidents expected);

XX, low-to-moderate (marginal to inadequate margin of safety; 6-7 accidents expected);

XXX, moderate (10-12 accidents expected; death possible);

XXXX, moderate-to-high (20-25 accidents expected; death likely).

*These risks are common to all remedial options and are expected to be minimized by a safety plan (see text); they are not included in the overall characterization, which classifies incremental risks associated with each option.

APPENDIX A
MATERIALS REVIEWED FOR THIS REPORT

Final Analytical Reports completed by Environmental Testing and Certification Corporation of Edison, New Jersey

Final Summary Report of Phase I
(Initial Audit for PCBs)
March 23-25, 1983
Addendum--Special Sampling Split with USEPA for Skim Oil Tank and Tank W-2

Final Summary Report of Phase II
(USEPA Split)
March 29-30, 1983

Final Summary Report of Phase III
(On-site Soil and Surface Water Monitoring for PCBs)
April 7-8, 1983

Final Summary Report of Phase IV
(On-site Ground Water Monitoring for PCBs)
April 18-21, 1983

Final Summary Report of Phase VI
(Off-site Surface Water Monitoring, Racoon Creek, for PCBs)
April 29, 1983

Final Summary Report of Phase VIII
(Additional Surface Water Monitoring for PCBs and Volatiles of Open Ponds 12, 11, 7, 5 and 4)

Draft Summary Reports completed by Environmental Testing and Certification Corporation of Edison, New Jersey

Draft Summary Report Phase Va
(Open Pond Sludge Characterization for PCB, Dioxin, and E.P. Toxicity on Ponds 12, 11, 7, 5, and 4)
April 25--May 6, 1983

Chemical Waste Management, Inc.

Air Sampling Results (personal air samples for PCBs taken during remedial work, May-June 1983)

Presentation to Ohio EPA on July 28, 1983,
Attachment B:

A. CWM Proposal for Remedial Action and Environmental Control Upgrading.

B. Exhibit I--Analytical data from ETC re: Sludge PCB Analysis.

C. Exhibit II--Sludge Remedial Operations.

D. Exhibit III--Site Integrity.

E. Exhibit IV--RCRA Landfill.

F. Exhibit V--Draft Consent Decree.

Reports and Letters from Environmental Research and Technology, Inc.

Draft Report: ERT Document No. B884-100
(Ambient PCB Testing at the CWM Facility in Vickery, Ohio)
April 1983

Letter from ERT to G.R.O.W.S., Inc., Reclamation
(Results of Limited Personal Monitoring for PCBs at Vickery; with Accompanying Table)
May 26, 1983

Draft Report: ERT Document B891-100(A)
(Preliminary Engineering Estimates of Volatile Organic Compounds and Inorganic Acids from Active Ponds, Vickery, Ohio)
June 1983

ERT Document No. B959-400-B
(Air Monitoring for PCBs and Inorganic Acids at CWM, Vickery, Ohio)
July 1983

Letter from ERT to CWM
(Impact of Revised Priority Pollutant Concentrations on Engineering Estimates of Pond Emissions; with Accompanying Table)
July 12, 1983

Letter from ERT to Clement Associates, Inc.
(Meteorological data for Toledo, Ohio)
July 22, 1983

Allied Analytical and Research Laboratories

Analytical Report No. 59513, "Pond 5"
Priority Pollutant Sampling Report
July 23, 1982

Analytical Report No. 59513, "Pond 7"
Priority Pollutant Sampling Report
July 23, 1982

Analytical Report No. 59780, "Pond 12"
Priority Pollutant Sampling Report
August 11, 1982

Golder Associates

Report on Geotechnical Data Review, Vickery, Ohio
Chemical Waste Management Facility
June 30, 1983

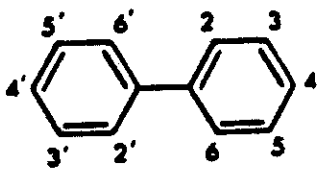
APPENDIX B

SUMMARY OF THE TOXICITY OF POLYCHLORINATED BIPHENYLS

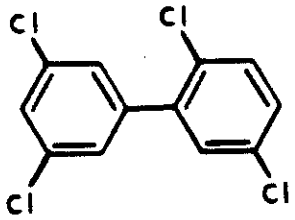
This APPENDIX summarizes the scientific information that is most relevant to the assessment of the possible risks posed by exposure to low levels of PCBs in the environment. It is divided into three parts. The first summarizes the chemistry of PCBs and described the nature of the commercial mixtures, including the presence of highly toxic trace contaminants. The second summarizes the most relevant information on the toxicity of PCBs, using information both from epidemiological studies and from experimental studies in laboratory animals. The third summarizes the available information on the toxicity of the toxic contaminants. Together these sections summarize the most important information on the known effects of PCBs, illustrate the complexity of the problem of risk assessment, and document the statements made in the text and in Appendix D.

I. Nature and Composition of PCB Mixtures

Commercial polychlorinated biphenyls (PCBs) are complex mixtures of chlorobiphenyls with varying degrees of chlorination. As indicated below, the parent biphenyl molecule has ten positions at which chlorine substitution can occur.



When the number but not the location of chlorine substitutions in a particular chlorobiphenyl is known, the chlorobiphenyl is referred to as a "congener" or "homolog" and is designated by a numerical prefix, e.g., tetrachlorobiphenyl. When the exact location of the chlorine substituents is known, the substance is referred to as an "isomer" and is identified by means of the numbering system indicated in the figure above. For example



is 2,3',5,5'-tetrachlorobiphenyl, one of 42 possible specific isomers of tetrachlorobiphenyl. Altogether there are 209 possible chlorobiphenyl isomers, at least 70 of which have been identified in commercial PCB mixtures.

Commercial production of PCBs involved chlorination of biphenyl with anhydrous chlorine in the presence of a catalyst which may be iron filings or ferric chloride, followed by treatment with alkali and distillation to purify the product. Depending on the process conditions, the product was a more or less complex mixture of chlorobiphenyls which was generally identified by the approximate percentage of chlorine it contained. In the case of the Aroclors formerly produced by Monsanto, the last two digits of the numerical designation (except for Aroclor 1016) indicated the approximate chlorine content; for example

Aroclor 1254 contained about 54% chlorine and Aroclor 1242 contained about 42% chlorine. Other PCB manufacturers used different designations; for example the French Phenoclor DP6, and the German Clophen A60, like Aroclor 1260, contained about 60% chlorine. Other commercial PCB mixtures included the Kanechlors formerly produced in Japan and the Fenclors formerly produced in Italy.

The molecular composition of several different PCB mixtures has been investigated in several studies (see IARC 1978). In one such study by Webb and McCall (1973), the approximate composition of several Aroclors was determined as indicated in Table B.1. There is evidently considerable overlap in composition between different Aroclor mixtures.

In addition to chlorobiphenyls, commercial PCBs may contain small quantities of impurities. Of particular importance are the highly toxic chlorinated dibenzofurans (CDFs) first identified as impurities in PCBs by Vos et al. (1970). CDFs have been detected at levels of 1-2 ppm in several Aroclors (Bowes et al. 1975a), at up to 13.6 ppm in Phenoclor DP-6 (Bowes et al. 1975a), and at up to 18 ppm in Kanechlor 400 (Nagayama et al. 1976). In a more recent study by Morita et al. (1977), 16 different Japanese and European PCB mixtures were analyzed and found to contain 1-16 ppm of CDFs (Table B.2). Of particular interest was the finding that a sample of a Japanese PCB mixture (Aroclor T1248) contained 2.8 ppm CDFs while Aroclor T1248 which had been used for 2 years in a heat exchange unit had

TABLE B.1

PERCENTAGE OF EACH CONGENER IN SEVERAL AROCLORS

Number of Chlorines	Aroclor					
	1221	1232	1242	1248	1254	1260
0	7	6				
1	51	26	1			
2	38	29	17	1		
3	3	24	40	23		
4		15	32	50	16	
5		0.5	10	20	60	12
6			0.5	1	23	46
7					1	35
8						6
9						
10						

TABLE B.2

CONCENTRATIONS (ppm) OF CDFs
OF DIFFERENT DEGREES OF CHLORINATION
IN PCB SAMPLES AND "YUSHO OIL"

PCB	3-Cl	4-Cl	5-Cl	6-Cl	Total	Reference
Aroclor 1248	--	0.5	1.2	0.3	2.0	1
Aroclor 1254[a]	--	0.1	0.2	1.4	1.7	1
Aroclor 1254[b]	--	0.2	0.4	0.9	1.5	1
Aroclor 1260[a]	--	0.1	0.4	0.5	1.0	1
Aroclor 1260[b]	--	0.2	0.3	0.3	0.8	1
Aroclor 1016	--	<0.001	<0.001	<0.001	--	1
Aroclor T64	--	4.8	9.4	2.0	16.2	2
Aroclor T241	--	2.4	2.7	0.8	5.9	2
Aroclor T1242	--	2.3	2.2	--	4.5	2
Aroclor T1248	--	0.5	2.3	--	2.8	2
Aroclor T1248[c]	0.3	5.8	5.6	0.7	12.4	2
Aroclor T1254	--	0.1	3.6	1.9	5.6	2
Aroclor T1260	--	0.8	0.9	0.5	2.2	2
Kanechlor KC300	--	6.7	1.6	--	8.3	2
Kanechlor KC400	0.3	12.2	10.4	0.9	23.8	2
Kanechlor KC500	0.2	1.7	1.1	3.1	6.1	2
Kanechlor KC600	--	0.2	0.5	0.4	1.1	2

TABLE B.2, (continued)

PCB	3-Cl	4-Cl	5-Cl	6-Cl	Total	Reference
Clophen A-30	1.6	2.3	1.0	--	4.9	2
Clophen A-40	1.5	5.4	6.9	--	13.8	2
Clophen A-50	0.7	8.3	4.1	1.8	14.9	2
Phenoclor DP-4	--	1.7	1.6	0.5	3.8	2
Phenoclor DP-5	--	4.6	2.7	2.6	9.9	2
Phenoclor DP-6[a]	0.2	2.1	2.6	5.6	10.5	2
Phenoclor DP-6[b]	--	0.7	10.0	2.9	13.6	1
Yusho oil	0.02	0.5	1.3	0.8	2.7	2

[a,b]Different samples

[c]Used PCB (2 years in heat exchanger)

SOURCE: Bowes et al. 1975a, Morita et al. 1977

12.4 ppm CDFs. Buser et al. (1978a) analyzed the same used PCB mixture and found slightly higher levels of CDFs (15.6 ppm).

High levels of CDFs were also found in the contaminated rice oil responsible for the disease known as Yusho. This disease occurred in Western Japan in 1968 among individuals who had consumed rice oil which had been contaminated by PCBs from a leaking heat exchanger. Estimates of the amount of CDFs in the "Yusho oil" have ranged from 2.68 ppm (Morita et al. 1977) to 5 ppm (Nagayama et al. 1976) and 5.6 ppm (Buser et al. 1978a). This corresponds to about 0.5% of the amount of PCBs in the Yusho oil. The high levels of CDFs in used PCBs may be the result of thermal decomposition of the PCB since CDFs are formed if PCBs are heated in air at temperatures above 270°C (Buser et al. 1978b, Morita et al. 1978). In addition to the PCBs and CDFs, the rice oil responsible for the Yusho incident also contained over 800 ppm of polychlorinated quaterphenyls (PCQs) (Miyata et al. 1978); polychlorinated quaterphenyl ethers (PCQEs) have also been identified in the rice oil (Miyata et al. 1979). Like CDFs, PCQs are formed when PCBs are heated, and up to 31,000 ppm of PCQs has been found in used PCB mixtures (Miyata et al. 1978). Very little is known about the toxicity of PCQs.

The presence of CDFs in PCB mixtures complicates interpretation of toxicology studies since CDFs are generally more toxic than PCBs (see Section III). However, the presence of CDFs

cannot be ignored, particularly if the finding of higher levels of CDFs in used than in unused PCBs is of general applicability.

II. Toxicity of PCBs

The toxicology of PCBs has been extensively reviewed in the past. Appendix C lists 25 reviews published since 1972. The following description of major toxicological findings is based upon these reviews, but emphasizes recent important studies.

A. Chloracne and Yusho

Current knowledge of the effects of PCB exposure in humans comes largely from cases of accidental contamination of food by PCBs and from cases of occupational exposure to PCBs. The effects of occupational exposure to PCBs have been reviewed in detail by NIOSH (1977). The classic symptom of occupational PCB exposure is chloracne, a disfiguring skin disease characterized by small dermal cysts, pustules and comedones, most commonly on the face, ears, and neck, but also on areas in contact with contaminated clothing. However, chloracne is a symptom of systemic poisoning and not just the result of skin contact since symptoms of chloracne occurred in cases of eating contaminated food in the Yusho incident in Japan (Kuratsune et al. 1972) and in a similar case in Taiwan (Chang et al. 1980a, 1980b, 1981). NIOSH (1977) concluded that chloracne may occur in individuals exposed to PCB vapor concentrations as low as 0.1 mg/m^3 for several months, or with blood PCB levels of about 200 ppb or more. Other effects that have been attributed to

occupational exposure to PCBs include digestive disturbances, eye irritation, liver injury, and impotence (NIOSH 1977).

More recently, Warshaw et al. (1979) reported an unusual incidence of abnormalities in pulmonary function in a group of capacitor-manufacturing workers exposed to PCBs. Among 243 workers with no history of exposure to asbestos, talc, or textile fibers which might affect pulmonary function, 34 (14%) showed impaired forced vital capacity (FVC). Impairment was defined as an age- and sex-specific FVC less than 79.5% of that predicted by standard values for healthy nonsmoking adults.

The incidence of impaired FVC among the capacitor workers (34/243, 14%) was similar to that seen by the authors among asbestos insulation workers (22/182, 12.1%) and greater than among nonsmoking adults (54/957, 5.5%). Among the 34 workers with impaired FVC, 27 had restrictive impairment but only one of these had radiographic abnormalities on a chest X-ray. The authors noted that restrictive impairment without radiographic changes is unusual in occupational exposures and that a reduction in FVC is not a characteristic change in cigarette smokers except when accompanied by generalized airway obstruction, rather than restriction. This finding of impaired pulmonary function among workers exposed to PCBs deserves further study.

Alvares et al. (1977) found that the plasma half-life of aminopyrine in five workers exposed to Aroclor 1016 for 2 years and other Aroclors prior to that was shorter than the

plasma half-life in a control group. Since aminopyrine is metabolized by the hepatic mixed function oxidase (MFO) system, the shorter half-life indicates these hepatic enzymes were induced in the exposed workers.

PCBs present in human mothers are transferred to babies via the milk (Kuwabara et al. 1978). Mothers (N=20) occupationally exposed to Kanechlor 300 and/or Kanechlor 500 had PCB blood levels of 8.3 to 84.5 ppb. while their children (N=39) had blood levels of 0.8 to 93.2 ppb. The PCB levels in the children aged 0-13 years were directly related to the length of time each was nursed. Infants not breast-fed by exposed mothers had PCB blood levels lower than their mothers'. Gas chromatographic analysis of mother and child's blood showed the same general peak pattern, but the peaks were smaller in the children. The maternal exposure or absorption of PCB was not quantified.

Two major incidents of dietary exposure to PCBs have been reported. The first occurred in 1968 in western Japan. The initial investigations of this disease have been reviewed by Kuratsune et al. (1972). The symptoms first reported were a chloracne-like skin disease which was given the name Yusho (oil disease) when the cause was identified as a particular brand of rice oil that had been contaminated during manufacture by a leaking heat exchanger. Recent estimates indicate that the most contaminated batch of rice oil contained about 1000 ppm PCBs, 5 ppm CDFs, and 866 ppm PCQs (Hayabuchi et al. 1979);

the presence of PCQEs has also been reported (Miyata et al. 1979). More than 1,000 individuals were affected by the disease. Common symptoms are indicated in Table B.3 which is taken from Kuratsune et al. (1972).

A dose-response relationship was noted between the amount of contaminated oil used and the severity of the disease as indicated in Table B.4 (Kuratsune 1972). More recently Hayabuchi et al. (1979) estimated the oil consumption of 72 Yusho victims and determined that the severity of the symptoms correlated well with the total oil consumption but not with the daily consumption on a μl/kg/day basis. These authors estimated that the average total amounts of PCBs, CDFs, and PCQs consumed by Yusho victims were 466, 2.5, and 439 mg, respectively, while the smallest total amounts consumed by symptomatic individuals were 111, 0.6, and 105 mg respectively, corresponding to daily doses of 29, 0.16, and 27 μg/kg/day, respectively.

Symptoms of the disease were seen in babies born to women exposed to PCBs either during pregnancy or during breast feeding. The babies exposed in utero had grayish, dark-brown stained skin at birth, most had increased eye discharge, and several were small-for-date. One baby born before its mother was exposed apparently contracted the disease subsequently through breast feeding (Kuratsune et al. 1976). One stillborn fetus showed marked hyperkeratosis and atrophy of the epidermis, and systic dilation of hair follicles, especially on the head (Kuratsune et al. 1972).

TABLE B.3

DISTRIBUTION OF SYMPTOMS REPORTED BY 189 YUSHO VICTIMS EXAMINED BEFORE OCTOBER 31, 1968

Symptoms	Females (N=89) (%)	Males (N=100) (%)
Dark brown pigmentation of nails	83.1	75.0
Distinctive hair follicles	64.0	56.0
Increased sweating at palms	50.6	55.0
Acnelike skin eruptions	87.6	82.0
Red plaques on limbs	20.2	16.0
Itching	42.7	52.0
Pigmentation of skin	75.3	72.0
Swelling of limbs	20.2	41.0
Stiffened soles in feet and palms of hands	24.7	29.0
Pigmented mucous membrane	56.2	47.0
Increased eye discharge	88.8	83.0
Hyperemia of conjuctiva	70.8	71.0
Transient visual disturbance	56.2	55.0
Jaundice	11.2	11.0
Swelling of upper eyelids	71.9	74.0
Feeling of weakness	58.4	52.0
Numbness in limbs	32.6	39.0
Fever	16.9	19.0
Hearing difficulties	18.0	19.0
Spasm of limbs	7.9	8.0
Headache	30.3	39.0
Vomiting	23.6	28.0
Diarrhea	19.1	17.0

TABLE B.4

RELATION BETWEEN AMOUNT OF RICE OIL USED AND CLINICAL SEVERITY OF YUSHO

Amount of oil used	Unaffected		Light cases		Severe cases		Total	
	No.	(%)	No.	(%)	No.	(%)	No.	(%)
Less than 720 ml	10	(12)	39	(49)	31	(39)	80	(100)
720-1440 ml	0	(0)	14	(31)	31	(69)	45	(100)
More than 1440 ml	0	(0)	3	(14)	18	(86)	21	(100)

These authors also reported that after exposure had ceased, during the period between summer 1969 and 1970, of 159 patients examined 81 had improved, 20 had worsened, and 58 had remained unchanged, and that even among those who had improved many still had serious complaints including persistent headache, general fatigue, weakness, numbness of the limbs, and weight loss (Kuratsune et al. 1972). Some patients displayed a peripheral neuropathy that was selective for sensory nerves (Murai and Kuroiwa 1971). By 1974, the skin symptoms had generally improved but symptoms such as general fatigue, poor appetite, headache, numbness and pain in the legs, cough and expectoration of sputum continued to be prominent for at least 10 years after exposure (Kuratsune et al. 1976, Urabe et al. 1979). Kuratsune et al. (1976) also reported a high incidence of menstrual disorders among Yusho

victims in 1974. The respiratory symptoms (cough and expectoration) were frequently associated with shadows on chest X-rays, and among 12 patients with respiratory distress who were studied in detail, 5 had chronic viral or bacterial airway infection suggesting reduced resistance to infection, possibly associated with the known effects of PCBs on the immune system (Shigematsu et al. 1978). The affected babies born to mothers exposed during pregnancy improved as they grew older (Kuratsune et al. 1972, 1976) and have not displayed any neurobehavioral effects as have been seen in monkeys and mice exposed during gestation and via breast milk.

The second major incident of contamination of food with PCBs also affected more than 1,000 persons and occurred under similar circumstances in central western Taiwan in 1979 (Chang et al. 1980 a,b, 1981). Once again contamination of rice bran oil by PCBs was responsible and a similar spectrum of symptoms was noted; acne-like skin eruptions, pigmentation of the nails, swelling of the eyelids and increased eye discharge (hypersecretion of Meibomian gland), headache, nausea, and numbness of the limbs. In addition to the effects seen in the Yusho incident, liver damage, immunologic impairment, and increased urinary excretion of heme precursors were noted. These effects had previously been observed in animals (see below). Liver damage was indicated by increased activities of the enzymes SGOT, SGPT, and alkaline phosphatase in blood serum (Chang et al. 1980a). The individuals exposed to PCBs had lower serum

γ-globulin levels and displayed decreased delayed-type-hypersensitivity responses to streptokinase and streptodornase (Chang et al. 1980a). These individuals also had decreased levels of immunoglobulins IgA and IgM in their serum, and lower percentages of T cells, especially helper T cells, among their lymphocytes (Chang et al. 1981). Chang et al. (1980b) examined the urinary excretion of heme precursors in 69 patients exposed to PCBs and 20 normal individuals. Significantly increased levels of δ-aminolevulinic acid and uroporphyrin were noted in the 24-hour urine of the PCB-exposed individuals compared to the normal subjects. No effect on urinary excretion of porphobilinogen or coproporphyrin was noted. Reports of the Taiwan incident have not mentioned CDFs, PCQs, or other contaminants.

In a recent epidemiological study of 458 members of a community in Alabama with no unusual exposure to PCBs, except that from a high intake of fish caught locally, a significant ($p<0.05$) positive correlation between serum PCB level (range 3.2-157.4 μg/liter, geometric mean 17.2 μg/liter) and diastolic blood pressure in adults was noted (Kreiss et al. 1981). Positive correlations were also observed between serum PCB concentration and gamma-glutamyl transpeptidase (GGTP) serum level, an indicator of liver disfunction, and between PCB level and serum cholesterol level. This study is of particular importance since it suggests the occurrence of subtle adverse effects resulting from exposure to relatively low ambient levels of PCBs. In a study of a smaller number of individuals (148)

with varying degrees of exposure to PCBs, Baker et al. (1980) also observed a correlation between serum PCB and GGTP levels; however, this correlation was eliminated when alcohol drinkers were excluded from the analysis. Baker et al. also noted a positive correlation between PCB level and serum triglyceride level as had been seen in Yusho victims in Japan (Kuratsune 1972); this correlation remained significant when analysis was limited to nondrinkers. However, it is not clear whether these associations between PCB levels in the blood and indicators of cardiovascular risks would indicate a cause-and-effect relationship. Since PCBs are associated with lipids in the blood, it is possible that individuals with elevated serum triglycerides would have elevated PCB levels for that reason.

B. Evidence of Carcinogenicity in Humans

There is no direct evidence that PCBs are carcinogenic in humans. However, Unger and Olsen (1980) have noted a statistically significantly ($p<0.01$) greater concentration of PCBs and DDE (a metabolite of the insecticide DDT) in adipose tissue of terminal cancer patients with a wide variety of types of cancer, compared to that of patients who died of other diseases, when the statistics were adjusted for age and sex. Also, Wassermann et al. (1978) observed higher levels of PCBs in the tissues of patients with gastric carcinoma than in the controls ($p<0.05$); and within the tissues of the cancer patients, the level of PCBs was higher in the tumor tissue than in adjacent normal

gastric mucosa ($p<0.01$). However, similar associations were observed with DDE and other organochlorines, and these findings are therefore ambiguous.

In a retrospective cohort mortality study of PCB-exposed workers making electrical capacitors (Brown and Jones 1981), mortality for all causes was lower than expected on the basis of U.S. age- and sex-adjusted, cause-specific mortality rates (163 observed, 182.4 expected). Mortality from all types of cancer combined was also lower than expected (39 observed, 43.8 expected). There was, however, a slight but nonsignificant excess of rectal cancer (4 observed, 1.19 expected) and liver cancer (3 observed, 1.07 expected), and in one plant there was a slight excess of deaths due to cirrhosis of the liver (5 observed, 3.2 expected). The latter two effects may be consistent with the known hepatotoxic effects of PCB, but the numbers involved are too small for any reliable conclusions to be drawn.

Bahn et al. (1976) reported an excess of melanomas (2 observed, 0.04 expected) among 31 men previously exposed to PCB in the research laboratory of a petrochemical plant. However, the extent of exposure to other potential carcinogens was not reported. Hence the cause of the melanomas is not clear.

A somewhat elevated incidence of death due to cancer has also been noted among Yusho victims. Among 31 deaths whose causes were confirmed up to 1977, 11 were from neoplasms (35.4% of the total). This fraction is higher than that observed

(21.1%) for deaths due to neoplasms among inhabitants of the same area of Japan in 1977 (Urabe et al. 1979). However, this comparison was not standardized for age or other differences between the two populations. Among the cancer deaths were 2 with stomach cancer, 1 with both stomach and liver cancer, 2 with liver cancer and liver cirrhosis, and 3 with tumors of the lung.

C. Effects Observed in Experimental Animals

Many of the effects of PCB exposure seen in humans are also observed in animals, and in many cases these effects can be studied more easily in animals than in humans. In addition, there are effects seen in animals that may potentially occur in humans; but evidence for their occurrence in humans is lacking, especially carcinogenicity and mutagenicity. The major effects of repeated exposure of animals to PCBs are briefly summarized below.

1. Carcinogenicity

There is evidence from several studies to implicate PCBs in the production of liver tumors in rats and mice, and there is some evidence to suggest that PCBs may also induce tumors of the stomach and lower gastrointestinal tract in rats.

Kimbrough et al. (1975) fed Aroclor 1260 at 100 ppm in the diet of female Sherman rats for 21 months. Among the 184 surviving treated rats, 26 had hepatocellular carcinoma and an additional 144 had neoplastic nodules in the liver. Among

173 surviving control animals, only 1 had liver carcinoma and none had neoplastic nodules.

The National Cancer Institute (1978) performed a carcinogenesis bioassay on Aroclor 1254. Groups of 24 male and 24 female Fisher 344 rats were fed diets containing 0, 25, 50, and 100 ppm Aroclor 1254 for 104-105 weeks. There was a small, dose-related increase in the incidence of liver hepatocellular adenoma and carcinoma and a larger increase in the incidence of nonneoplastic liver nodular hyperplasia as shown in Table B.5 below.

TABLE B.5

INCIDENCE OF LIVER LESIONS IN RATS IN NCI BIOASSAY

	Control	25 ppm	50 ppm	100 ppm
Hepatocellular adenoma and carcinoma (males)	0/24	0/24	1/24	3/24
Hepatocellular adenoma and carcinoma (females)	0/23	0/24	1/22	2/24
Nodular hyperplasia (males)	0/24	5/24	8/24	12/24
Nodular hyperplasia (females)	0/23	6/24	9/22	17/24

If males and females are combined, the incidence of hepatocellular adenoma and carcinoma is significantly greater in the 100 ppm group than in the control group ($p=0.030$, Fisher's exact test). In addition to these liver lesions, there was

a total of four adenocarcinomas and one carcinoma in the gastrointestinal tract of treated rats but none in the controls. These are uncommon tumors in this strain of rats. The historical incidence of such tumors is 6/600 in males and 2/600 in females. NCI concluded that these tumors may have been related to the Aroclor 1254 treatment. The incidence of hepatocellular adenoma and carcinoma seen at 100 ppm in this study (10.4%) is consistent with the incidence (14.1%) observed in the study by Kimbrough et al. (1975), but the high incidence of neoplastic nodules seen in the Kimbrough et al. (1975) study was not seen in the NCI bioassay. A high incidence of nodular hyperplasia was seen, however. Nodular hyperplasia is considered an early, preneoplastic lesion which may progess to neoplasia under suitable circumstances (Pitot and Sirica 1980). Thus the main difference between the results of the two experiments is that the tumors developed more slowly in the NCI study.

Kimbrough and Linder (1974) also reported that Aroclor 1254 fed to BALB/cJ male mice at 300 ppm in the diet for 11 months induced hepatomas in 9/22 surviving animals. No hepatomas were seen in the livers of 58 mice fed a normal diet for 11 months. The PCB-fed mice also had greatly enlarged livers and adenofibrosis of the liver.

2. Interactions with Other Carcinogens

Early studies of interaction indicated that treatment of animals with certain carcinogens and with PCBs at the same time delayed or inhibited the action of the carcinogen. For

example, Makiura et al. (1974) observed a greatly reduced incidence of liver tumors in rats fed PCB at the same time as 3'-methyl-4-dimethylaminoazobenzene (3'-Me-DAB), N-2-fluorenylacetamide (2-FAA) or diethylnitrosamine (DENA). The authors speculated that the inhibitory effects may be attributed to stimulation of microsomal enzymes by PCB, enhancing metabolism of the carcinogens.

More recently, several studies have indicated that when PCBs are administered to rats following a low dose of a liver carcinogen, the incidence of liver tumors is greatly enhanced by the PCB treatment. This promoting effect has been observed with DENA (Nishizumi 1976, 1979, Preston et al. 1981), 2-FAA (Ito et al. 1978, Tatematsu et al. 1979), and 3'-Me-DAB (Kimura et al. 1976). In the study by Preston et al. (1981), for example, groups of 40 male Sprague-Dawley rats were given drinking water containing DENA at 66 µg/ml for 5 weeks, and then Aroclor 1254 was added to the diet of one group for a further 18 weeks. An additional group of 40 did not receive DENA but were given Aroclor 1254 in weeks 6-23. An untreated group was also included. In rats surviving to 23 weeks, none of the control rats or rats receiving only Aroclor had liver tumors. Of 32 rats receiving only DENA, 5 (16%) had hepatocellular carcinomas, while 21/33 (64%) of the rats given DENA followed by Aroclor had hepatocellular carcinomas. In the study by Kimura et al. (1976), Kanechlor 400 at 400 ppm in the diet for 6 months enhanced the production of hepatocellular carcinoma in rats previously fed 3'-Me-DAB at 600 ppm in the diet for 2 months. However,

the incidence of carcinoma was reduced if Kanechlor was given before or concurrently with 3'-Me-DAB. Also, when rats were exposed in utero and via their mothers' milk to PCBs (Kanechlor 500), they were less sensitive than unexposed rats to subsequent induction of liver tumors by DENA (50 ppm for 5 weeks after weaning), showing a significantly reduced ($p<0.05$) number of liver tumors per rat (1 tumor/rat in males, 0 in females) compared to rats not exposed to Kanechlor (3 tumors/rat in males, 1.1 in females) (Nishizumi 1980). The fact that increased tumor yield occurs only when PCBs are given after the carcinogen suggests that the effect is not simply related to the ability of PCBs to induce liver enzyme systems and increase metabolic activation of the carcinogen. Rather, PCBs appear to be acting as promoters. However, PCBs neither have promoting activity in the classical two-stage mouse skin tumorigenesis system using 7,12-dimethylbenz(a)anthracene as the initiator (Berry et al. 1978), nor in a system in which the cervical epithelium of mice was treated with 3-methylcholanthrene (MC) for 4 weeks and the mice given a diet containing 10 or 100 ppm Kanechlor 400 during and for 3 weeks after MC treatment (Uchiyama et al. 1974). However, these authors did not try administering PCBs only after treatment with the carcinogen, but used concurrent treatments.

3. Mutagenicity

Most studies of the mutagenicity of PCBs have given negative results, including assays for reversion in Salmonella typhimurium

(Hsia et al. 1978, Schoeny et al. 1979), chromosome aberrations in rat bone marrow (Green et al. 1975a), dominant lethal mutation in rats (Green et al. 1975b), and chromosome breakage in Drosophila (Nilsson and Ramel 1974). These studies used 2,2',5,5'-tetrachlorobiphenyl, Aroclors 1242 and 1254, and Clophens A30 and A50. However, another study has provided evidence that chlorinated biphenyls with a lower degree of chlorination may be mutagenic in Salmonella strain TA1538 in the presence of a rat liver metabolic activation system (Wyndham et al. 1976). Wyndham et al. (1976) also noted binding of ^{3}H-4-chlorobiphenyl to protein and RNA when the chemical was added to a liver microsome preparation. In a later study, Wyndham and Safe (1978) presented evidence that 4-chlorobiphenyl was metabolized via an arene oxide intermediate. Arene oxide intermediates are the reactive derivatives believed to be involved in the binding of carcinogenic polynuclear aromatic hydrocarbons to DNA. It is likely that the arene oxide intermediate is responsible for the mutagenic activity of 4-chlorobiphenyl. Negative results were obtained with 4-chlorobiphenyl in a modified assay for mutagenicity in Salmonella (McMahon et al. 1979), but it is unclear what doses were tested or how the modifications used may have affected the sensitivity of the assay.

4. Reproductive, Teratogenic, and Postnatal Effects

Exposure to PCBs at various times before, during and after pregnancy has resulted in various degrees and types of adverse effects on reproduction and on the offspring in many species.

Among the species most sensitive to these effects of PCBs are the monkey and the mink, but all species that have been tested, including rats, mice, rabbits, and miniature swine, have shown effects. Studies with monkeys (Allen et al. 1979, 1980; Bowman et al. 1978, 1981) have examined the reproductive effects of PCBs and the redistribution of maternal PCBs to the fetus transplacentally and to the nursing offspring via the milk.

Adult female rhesus monkeys received Aroclor 1248 at dietary levels of 0.5, 1.0, 2.5 or 5.0 ppm for 18 months. Prolonged menses and elevated serum progesterone levels were observed after 4 months of treatment. The females were mated with untreated males after 7 months of treatment and again 1 year after PCB treatment was stopped. The results of mating for the females in the 2.5 and 5.0 ppm group are shown in Table B.6 below.

TABLE B.6

REPRODUCTIVE PERFORMANCE OF MONKEYS EXPOSED TO POLYCHLORINATED BIPHENYLS IN THEIR DIETS FOR 18 MONTHS

	During PCB Exposure		1 Year After PCB Exposure	
	2.5 ppm	5.0 ppm	2.5 ppm	5.0 ppm
Total impregnated	8/8	6/8	8/8	7/7
Absorptions/resorptions	3/8	4/8	1/8	1/7
Stillborn	0/8	1/8	0/8	1/7
Normal births	5/8	1/8	7/8	5/7

Infants in each treatment group in each mating had reduced body weights at birth. No gross external malformations were reported. After the first mating, the six infants were permitted to nurse for 4 months. Within 2 months focal areas of hyperpigmentation, swollen lips and eyelids, loss of eyelashes, and acneform lesions of the face developed in the infants. The skin of these infants showed a decided increase in the PCB level over this period. Within 4 months, three of the six infants died (one in the 5.0-ppm group and two in the 2.5-ppm group) due to PCB intoxication. After weaning, the remaining three showed improvement of the skin lesions during the subsequent 4-month period. At weaning and 2 years later, the surviving offspring of the first mating in the 0.5, 1.0, and 2.5-ppm groups were similarly hyperactive and had comparable deficits in learning ability. At 4 years of age these offspring were hypoactive (Bowman et al. 1981).

After the second mating, the breast milk of the mothers contained 0.02-0.19 μg PCB/g whole milk, and the hairline of the infants showed hyperpigmentation. Two infants in each group died after weaning. These four infants showed hypocellularity of the thymus, grossly small spleens, and reduced number of lymph nodes. Histologically the lymph nodes were hypocellular and devoid of germinal centers, while the bone marrow was hypoactive as evidenced by the majority of cells being mature (Allen et al. 1980). These histologic findings are indicative of

impairment of the immune system, an effect of PCBs which is discussed in more detail later.

Male monkeys appear to be less susceptible than females to reproductive dysfunction by PCBs (Allen and Norback 1976). Four adult male rhesus monkeys were given a diet containing 5.0 ppm Aroclor 1248 for 17 months (average total intake of PCBs 460 mg). They began to develop a slight periorbital edema after 6 months of exposure; however, it was much less severe than in the female monkeys receiving a similar level of PCB. The morphological features and viability of the spermatozoa, as well as the ability to fertilize control monkeys, was unaffected during the initial 12 months of PCB exposure. Subsequently, one of the four males lost weight and developed alopecia, acne, periorbital edema, and decreased libido. A testicular biopsy of this animal showed a decided hypoactivity of the seminiferous tubules. There was an absence of mature spermatozoa and a predominance of Sertoli cells of the tubules. The remaining three males remained healthy and sexually active (Allen and Norback 1976).

Reproduction in mink is also adversely affected by low levels of PCBs. The degree of reproductive impairment is different for different PCB mixtures. Aroclor 1254 at 2 ppm in the diet dramatically decreased the number of live kits born per female. Aroclor 1242 had a similar effect at 5 ppm in the diet. Aroclor 1016, however, had considerably less effect on reproduction. A dietary level of 2 ppm was without effect,

while 10 ppm reduced the number of successful pregnancies but did not affect the number of kits per litter (Aulerich and Ringer 1977, 1980, Bleavins et al. 1980).

The teratogenic potential of PCBs, both individual isomers and commercial mixtures, has been studied in several species. In a study by Masuda et al. (1979), mice were fed diets containing known levels of each of seven chlorobiphenyl isomers (tri- to octachloro) for 18 days either before or during pregnancy.

Several isomers reduced the number offspring per female, but the only isomer that caused measurable defects in the offspring was 3,3',4,4'-tetrachlorobiphenyl. Offspring of mice treated with this chemical showed increased locomotor activity and general restlessness ("waltzing syndrome") within 2-3 weeks of birth. It could not be determined whether placental transfer or uptake of PCB from maternal milk was the critical route of exposure (Lucier et al. 1978).

Tilson et al. (1979) and Marks et al. (1981) also reported neurobehavioral defects in the offspring of female mice treated with 3,3',4,4'-tetrachlorobiphenyl and 3,3',4,4',5,5'-hexachlorobiphenyl during pregnancy. However, these isomers are only minor components in commercial PCB mixtures, so the relevance of these studies to risk assessment for environmental PCBs is limited.

Several studies of the effects of PCB mixtures have also been conducted. Villeneuve et al. (1971) reported that single oral doses of 12.5, 25, and 50 mg/kg Aroclor 1254 administered

to pregnant rabbits were fetotoxic, but did not induce teratogenic effects. Linder et al. (1974) reported that Aroclors 1254 and 1260 had minor fetotoxic effects when administered at 100 mg/kg/day to female rats on days 7-15 of pregnancy; Aroclor 1254 also caused substantial mortality in pups prior to weaning.

Earl et al. (1974) reported that Aroclor 1254 caused reproductive impairment and skeletal abnormalities in the offspring of beagle dogs and miniature swine exposed during pregnancy to doses of 5 mg/kg/day and 10 mg/kg/day, respectively. However, this study was reported only in an abstract, and details are not available for review.

In summary, PCB mixtures have been reported to affect reproduction in a number of animal species, but rhesus monkeys and mink appear to be much more sensitive than other species. Teratogenic effects have not been convincingly reported, except for neurobehavioral effects in mice caused by isomers that are only very minor components in commercial mixtures.

5. Liver Toxicity

An increase in liver weight and/or in liver-to-body weight ratio in response to PCB treatment has been noted in many studies. Litterst et al. (1972) observed increased liver-to-body weight ratios in rats fed 50 and 500 ppm of Aroclors 1242, 1248, 1254, and 1260 for only 4 weeks. Similar increases in liver weight have been noted in long-term feeding studies with these mixtures in rats (Bitman et al. 1972; Linder et al. 1974). In addition,

effects were noted at much lower dietary levels in offspring of treated rats (exposed during gestation and prior to weaning): significant increases in liver weight were noted in weanling rats after maternal exposure to only 5 ppm Aroclor 1260 and only 1 ppm Aroclor 1254 (Linder et al. 1974).

Increase in liver weight has been noted as an effect of PCBs in many other species (Vos 1972, Nishizumi 1970, Barsotti and Allen 1975, Hansen et al. 1975).

The initial increase in liver weight is due mainly to proliferation of smooth surfaced membranes of the endoplasmic reticulum, a network of interconnected channels present in the cytoplasm of most animal cells. Norback and Allen (1972) observed a proliferation of smooth endoplasmic reticulum (SER) in rats fed PCBs for one to five weeks. Similar proliferation of SER has been observed in mouse and monkey livers (McNulty 1976, Nishizumi 1970).

Changes in biochemical composition of the liver after exposure to PCBs include a significant increase in total lipid content (Grant et al. 1971). Triglyceride content of the liver increased with increased dosage of PCBs (from 5 to 500 ppm) in the diet (Litterst et al. 1972). This increase was greater with Aroclor 1248 than with 1242, 1254, or 1260. Norback and Allen (1972) found the ratio of phospholipids to protein increased and the ratio of cholesterol to protein increased. Hinton et al. (1978), on the other hand, found that although the total amount of liver phospholipid and cholesterol increased, the

phospholipid/protein and cholesterol/protein ratios did not change when rats were treated with large doses of Aroclor 1254 (25 and 50 mg/kg). Both the total triglyceride and triglyceride/protein ratio increased markedly. They found the increase in phospholipids and triglycerides was caused by a decreased degradation or removal of the lipids from the liver and not an increase in synthesis. Marked increases in liver lipids have also been noted in treated rabbits (Ito et al. 1971).

6. Induction of Hepatic Mixed Function Oxidases

The hepatic mixed function oxidases (MFO) make up a membrane-bound enzyme system found mainly in the SER of the liver cells. MFO usually increases in activity with an increase in the SER. This enzyme system is able to metabolize and detoxify oxidatively a wide variety of exogenous and endogenous compounds. A major component of this system is a cytochrome enzyme, which because of its ability to absorb light, under specific conditions, with wavelengths around 450 nm, is called cytochrome P-450. Initially it was believed there was only one such enzyme, but it is now known that there is a family of these enzymes. Each cytochrome P-450 has its own enzymatic activities and spectral properties. In the normal animal not all the cytochrome P-450s are present. A variety of chemicals, such as PCBs, are, however, able to cause the synthesis of specific cytochromes and thus cause an increase in the total amount of enzyme and the enzymatic activity. This is considered induction of the MFO.

Disruptions in normal hepatic MFO activity have been observed with PCBs even at dose levels which had no effect on liver weight or on liver-to-body weight ratios (Litterst et al. 1972) Several hepatic enzyme systems are affected in rats at dietary levels as low as 5 ppm (Chen and Dubois 1973) or even 0.5 ppm (Litterst et al. 1972), and "no-effect levels" have not yet been found. Application of very small amounts of PCBs to the skin of experimental animals can cause a marked increase in MFO activity (Kappas and Alvares 1975).

After termination of exposure, the enzyme-inducing effect of PCBs appears to be reversible. Litterst and Van Loon (1974) maintained rats on a high dietary concentration of 50 ppm PCB for seven days. Discontinuation of PCB treatment resulted in a slow decay of the induced enzyme activity to approximately control levels after 10 days. The various effects of PCBs on liver microsomal enzymes can be blocked by the prior administration of actinomycin D (Kimbrough 1974). Actinomycin D acts as an inhibitor of DNA-dependent RNA synthesis. Alvares et al. (1973) interpreted the inhibitory effect of actinomycin D on PCB-induced increases in enzyme activity as support for the view that PCBs enhance the synthesis of a distinct microsomal hemo-protein not present in liver of untreated rats.

There are two general classes of inducers of MFOs. These classes are characterized by the compounds phenobarbital (PB) and 3-methylcholanthrene (3-MC). The two compounds induce different species of cytochrome P-450. Ryan et al. (1979)

isolated from rat liver the cytochrome P-450s induced by these compounds and by Aroclor 1254. Three cytochrome P-450s were isolated from treated rat liver and designated P-450a, P-450b, and P-450c. Phenobarbital induced P-450a and P-450c while 3-MC induced P-450a and P-450b. Molecular weight analysis, spectral properties, catalytic activity, reactivity with antibodies, and peptide mapping all showed that Aroclor 1254 induced three individual forms of P-450 and that they were identical to those induced by PB or 3-MC. Thus Aroclor 1254 has the characteristics of both PB and 3-MC for induction of MFOs.

A number of recent studies have started to elucidate structure-activity relationships in enzyme induction by chlorobiphenyls in rats (Ecobichon 1976, Poland and Glover 1977, Yoshimura et al. 1979, Goldstein 1979). The activity of PCB mixtures in inducing the specific enzymes characteristic of both PB and 3-MC type inducers has been shown to be caused by individual isomers that are either PB or 3-MC type inducers (Poland and Glover 1977, Goldstein et al. 1977, Goldstein 1979, Yoshimura et al. 1979). In the study by Yoshimura et al. (1979), the effect of individual isomers on the rat hepatic MFO activities of benzphetamine N-demethylase (BZ) and arylhydrocarbon hydroxylase (AHH) were measured as well as the concentration of cytochrome P-450. BZ activity, representative of PB type induction, was increased by specific PCB isomers, while AHH activity, representative of 3-MC type induction, was increased by other PCB isomers. The absorption spectrum of the cytochrome P-450

induced by the latter isomers was similar to that found with 3-MC induction, which causes a peak absorption at 448 nm, not 450 nm, and is thus referred to as cytochrome P-448. The absorption spectrum of the cytochrome P-450 induced by isomers with PB-type activity was similar to the PB-induced cytochrome P-450 spectrum with a peak absorption at 450 nm.

The toxicity of the individual isomers was also correlated to their type of MFO induction. The 3-MC type inducers were more toxic than PB type inducers. This toxicity, however, was not directly related to the induction of cytochrome P-448/P-450 (Yoshimura et al. 1979).

McKinney and Singh (1981) examined crystallographic, theoretical and toxicological data in developing an hypothesis on the structural specificity of PCB for 3-MC-type induction of cytochrome P-448. They found that the planar structure of PCBs with chlorines at the para positions of each ring and at least one meta position of each ring was very similar to the planar structure of 2,3,7,8-tetrachlorodibenzo-p-dioxin, a very potent 3-MC-type inducer. Such a structure can fit into a 3 x 10 Å rectangle. In addition to the ability to form this planar structure, relatively high polarizability of the molecule, which is determined by the number and position of the chlorines, is important for the molecule to have a high affinity to the receptor site responsible for MFO-induction.

of <0.1 µg/g. Aroclors 1016 and 1242 also induce porphyria in rats (Iverson et al. 1975, Goldstein et al. 1975). After dosing at 10 and 100 mg/kg/day for 21 days, both mixtures caused a significant accumulation of porphyrins in both sexes, but Aroclor 1242 was nearly twice as effective at 100 mg/kg and produced a significant effect at the lower dose.

Rabbits developed symptoms of porphyria after application of Clophen A50, Phenoclor DP6, and Aroclor 1260 to their back skin (Vos and Beems 1971).

8. Immunotoxicity

The ability of PCBs to inhibit immune responses has been reviewed by Vos (1977) and by Silkworth and Loose (1981). Treatment with PCBs has been shown to reduce the level of serum antibodies to pseudorabies virus in rabbits and to tetanus toxin in guinea pigs, reduce the delayed-type hypersensitivity response to tuberculin in rabbits and to Freund's complete adjuvant in guinea pigs, reduce the mixed lymphocyte response in guinea pigs, and increase the susceptibility of ducks to infection with duck hepatitis virus. PCBs also cause atrophy of the thymus and damage to the spleen in several species. Since the thymus and spleen are involved in immunologic reactions, these adverse effects are indicators of reduced immunocompetence.

More recently, Aroclor 1254 at 63, 135, 250 and 550 mg/kg has been shown to produce a dose-dependent reduction in plaque-forming cells in the spleen of mice in response to a challenge

7. Induction of Porphyria

Porphyria cutanea tarda in humans is an acquired defect in hepatic porphyrin metabolism, characterized by uroporphorinuria (excretion of porphyrins in the urine), photosensitivity as manifested by blisters, and mechanical fragility of the skin (Kimbrough 1974). Normally, porphyrin synthesis is well regulated to provide the necessary amount of porphyrins for heme synthesis. The hepatic porphyria which is responsible for an increase in porphyrins and for the skin photosensitivity can be produced experimentally by a number of drugs and chemicals which have the ability to stimulate activity of the initial enzyme of heme synthesis, δ-aminolevulinic acid (ALA)-synthetase (Strik and Wit 1972).

PCBs have been shown to cause hepatic porphyria in animals (Goldstein et al. 1974, Vos and Beems 1971). Goldstein et al. (1974) fed rats a diet containing 100 ppm Aroclor 1254 for up to 13 months. After 2-7 months, there was an increase in the urinary excretion of several intermediary compounds of the metabolic pathway for heme synthesis. Uroporphyrin excretion increased up to 540-fold while the excretion of coproporphyrin increased 27-fold, porphobilinogen increased 50-fold, and δ-ALA increased 18-fold. The activity of δ-ALA synthetase, the rate-limiting enzyme of the metabolic pathway, was increased after 3 months on the diet. At 4 months, the porphyrin concentration in the livers of treated rats was 1410 μg/g of liver compared to the concentration in the livers of control animals

with sheep red blood cells. This indicates that Aroclor 1254 inhibited the formation of antibody-producing cells in the spleen (Wierda et al. 1981). A similar effect was seen in Rhesus monkeys fed Aroclor 1248 at 5 ppm in the diet for 11 months. These animals had reduced hemolysin titers in response to injection with washed sheep red blood cells and reduced levels of γ-globulin in blood serum compared to control animals not exposed to PCBs (Thomas and Hinsdill 1978).

C. Toxicity of Chlorinated Dibenzofurans (CDFs)

As described earlier, commercial PCBs are contaminated with low levels of CDFs. CDFs may be formed from PCBs during heating and during use in heat exchangers, and high levels of CDFs were found in the rice oil responsible for the Yusho incident in Japan. CDFs have been detected in the adipose tissue and liver of Yusho victims at levels of about 0.01 ppm on a whole tissue basis (Kuratsune et al. 1976). Indeed they appear to be preferentially retained compared to PCBs since the ratio of PCB to CDF in the livers of the patients 7 years after exposure was about 4:1 compared to 200:1 in the contaminated rice oil.

The association of CDFs with PCBs and the apparent formation of CDFs from PCBs when heated are of particular concern because CDFs are considerably more toxic than PCBs. For example, in a comparative toxicity study, Oishi et al. (1978) examined the toxicity of a PCB mixture (Kanechlor 500) and a mixture of CDFs produced by chlorination of dibenzofuran. The CDF

mixture contained tetrachloro-, pentachloro-, and hexachloro-dibenzofurans (average number of chlorines per molecule, 4.7). Groups of 10 male Sprague-Dawley rats were fed for 4 weeks on diets containing 100 ppm PCB, 10 ppm CDF, or 1 ppm CDF; an additional group was given untreated feed. Body weight gains were less in the treated groups than in the control group. This effect increased in severity in the order 100 ppm PCB < 1 ppm CDF < 10 ppm CDF. The relative weights (g/100 g body weight) of brain, spleen, and liver were significantly increased in the animals fed 100 ppm PCBs. These effects were also produced by both dose levels of CDFs. In addition, both 1 ppm and 10 ppm CDFs significantly increased the relative weights of heart, lungs, adrenals, and testes, and significantly decreased the absolute and relative thymus weight. CDFs at 10 ppm also significantly decreased the relative weights of seminal vesicles and ventral prostate. Both doses of CDFs significantly reduced the blood hemoglobin concentration and hematocrit compared to the control group. This effect was not produced by the PCB treatment. All three treatments significantly increased the serum cholesterol concentration, significantly reduced the serum triglyceride concentration and significantly increased the concentration of lipid in the liver. It appears that 1 ppm CDF produced effects more severe than 100 ppm PCB in this study.

Although the toxicity of individual isomers of CDFs has not been studied in detail, emphasis has been placed on the isomer 2,3,7,8-tetrachlorodibenzofuran (2,3,7,8-TCDF) since

it is likely to be the most toxic by analogy with the structurally similar chlorodibenzo-p-dioxins (CDD), of which 2,3,7,8-TCDD is the most toxic (Poland and Glover 1973). 2,3,7,8-TCDF is also one of the isomers that is found in commercial PCBs (Bowes et al. 1975a, b), is formed during heating of PCBs (Morita et al. 1978), and is the major CDF in Yusho oil, in which it was present at 0.45 ppm, about 8% of the total CDFs (Buser et al. 1978a).

In rhesus monkeys, 2,3,7,8-TCDF at 5 and 50 ppb in the diet caused sickness and some deaths in groups of three animals fed for 6 and 2 months, respectively. The principal pathologic effects were atrophy or squamous metaplasia of the sebaceous glands, mucous metaplasia and hyperplasia of the gastric mucosa, involution of the thymus, and hypoplasia of the bone marrow. The effects were most severe at the 50 ppm dose level, but were also apparent at the lower dose level. The survivors recovered after 3 months on a TCDF-free diet (McNulty et al. 1981)

CDFs are also much more potent inducers of liver microsomal enzymes than PCBs. Kawano and Hiraga (1978) observed a greater increase in liver cytochrome P-450 content in rats given three daily doses of mixed CDFs at 100 µg/kg/day than in rats given three daily doses of a PCB mixture (Kanechlor 500) at 10 mg/kg/day. Also, Goldstein et al. (1978) observed that the highly toxic isomer 2,3,7,8-TCDF was active as an inducer of the liver microsome enzyme aryl hydrocarbon hydroxylase in rats when given

as three daily intraperitonal doses of 0.1 µg/kg/day. This was the lowest dose tested, and a no-effect level for this isomer has not been identified. Indeed, contamination of a commercial, 99% pure hexachlorobiphenyl isomer with 44 ppm 2,3,7,8-TCDF was responsible for the induction of cytochrome P-448 and aryl hydrocarbon hydroxylase by the commercial hexachlorobiphenyl (Goldstein et al. 1978). These results indicate that 2,3,7,8-TCDF was at least 20,000 times more potent as an inducer of AHH than 2,2',4,4',5,5'-hexachlorobiphenyl.

APPENDIX C

TOXICOLOGICAL DATA NOT USED IN THE RISK ASSESSMENT

In Section III of this report, we selected three types of toxic effect of PCBs as the basis for assessment of risks at low exposure levels. Chloracne is the best documented effect in humans and is an effect for which the exposure-response relationship has been defined reasonably clearly. Reproductive toxicity is the effect that has been observed in animal experiments at the lowest dose level. Carcinogenesis in animals is an effect of significant public health concern and an effect for which there are scientific reasons to believe that risks may exist at very low dose levels.

In addition to these types of toxic effect, several other effects of PCBs have been reported, but were not used in our risk assessment. The most important of these reported effects are listed below.

1. Data suggesting abnormal lung function in workers exposed to PCBs (Warshaw et al. 1979). In our judgment these findings need independent confirmation.

2. Data suggesting correlations between PCB levels in tissues and indicators of cardiovascular risks (Kreiss et al. 1981, Baker et al. 1980). Although these are potentially significant risk factors, it is not clear whether the elevated PCB levels associated with them are causes or effects.

3. Data on mutagenicity of lower chlorinated biphenyls (Wyndham et al. 1976) and on binding of metabolites to DNA

and RNA (Morales and Matthews 1979). The significance of these findings for risk assessment is not clear, since the mutagenic components are not found in commercial mixtures such as Aroclor 1254.

4. Data on the sensitivity of mink to low levels of PCBs. The validity of the mink as an experimental model for toxic effects in humans is questionable.

5. Data on teratogenic effects of PCBs. Studies by Earl et al. (1974) have not been reported in sufficient detail; Lucier et al. (1978) used 3,3',4,4'-tetrachlorobiphenyl, a highly toxic isomer that is only a minor component in commercial mixtures.

6. Data on induction of liver enzymes at low doses. Although several human studies have shown increased activity of certain liver enzymes (SGOT, SGPT, and GGP), the health significance of these findings is not clear. In theory, the increased activity of mixed function oxidases could either increase the risks of cancer (by augmenting the conversion of carcinogens into active metabolites), or decrease it (by accelerating the breakdown of these active metabolites). In practice, most studies have indicated a decrease in effects when PCBs are administered prior to administration of a carcinogen that requires metabolic activation (see Appendix B). Hence, we will assume that the net effect of enzyme induction by PCBs is likely to be beneficial or neutral.

APPENDIX D

POSSIBLE RISKS POSED BY TCDD

After the risk assessment of PCBs presented in this report was completed, trace quantities (up to 18 ppb) of 2,3,7,8-tetrachloro-p-dioxin (TCDD) were reported as having been identified in sludges from lagoons 4 and 5. In many respects, the biological effects of TCDD and PCBs are similar but TCDD is roughly 100,000 times more potent than PCB mixtures of the type found in sludges at the OLD site (Parkinson and Safe 1981). But, the intrinsic hazard posed by TCDD at concentrations of 5-18 ppb are somewhat greater than those posed by PCBs at concentrations around 100 ppm. However, this difference is at least partially offset by the fact that TCDD is less volatile than PCBs, is more strongly adsorbed to surfaces, and is less liable to move in groundwater. Hence the risk posed by TCDD at the level recorded at the OLD site are probably of the same order of magnitude as those posed by PCB. With appropriate changes, the conclusions drawn in this report about the risks posed by PCBs are equally applicable to TCDD.

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